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THE ROLE OF THE ROENTGENOLOGIC EXAMINATION IN THE DIAGNOSIS OF INTESTINAL OBSTRUCTION*

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PHILADELPHIA

IN studying patients who have symptoms suggestive of intestinal obstruction, some of the problems that the clinician must answer if possible include the following: Is an obstruction present? Does it involve the stomach, the duodenum, the small intestine, the colon or a combination of these organs? Is the obstruction single or multiple? Is the obstruction complete or incomplete? What is the cause of the obstruction? Is it an acute occlusion, or is it the final closure of a slowly developing lesion?

The most valuable aid on which the clinician may rely is probably the correct interpretation of observations gleaned from a carefully planned roentgenologic examination of the abdomen or the gastrointestinal tract. The discussion of such observations is limited, for the most part, to those concerning the small intestine and colon.

PRESENCE OF OBSTRUCTION

Patients with obstructive lesions in the intestinal tract, some of which are almost complete, do not always present classic symptoms. Occasionally, this is true of a lesion involving the pylorus and duodenum. In such cases, the diagnosis can be made only after the patient has had a roentgenologic study of the gastrointestinal tract.

As a rule, the patient with symptoms of an acute abdominal condition is referred to the radiologist for a survey examination of the abdomen. If the condition is such that he can be moved with safety, examination of the abdomen includes a fluoroscopic study in the horizontal and semierect positions. The advantage of such a procedure is to determine the extent of the movement of the domes of the diaphragm and to make, if necessary, roentgenograms of important fluoroscopic observations.

The minimum requisite of such a study is a roentgenogram of the abdomen in the anteroposterior position. Additional roentgenograms that may reveal valuable information are as follows: posteroanterior and lateral films with the patient in the recumbent position; anteroposterior or posteroanterior films with the patient lying on the left or right side; and anteroposterior or posteroanterior films with the patient in the erect position. Whenever possible, the shadows of the domes of the diaphragm should be seen on the roentgenogram, and a lateral view of the pelvic region should be available for study of gas shadows in the rectum.

The purpose of the survey is to determine the presence and location of any demonstrable gas in the intestinal tract or abdomen, the presence or absence of muscle shadows and the preperitoneal fat lines, and to locate if possible any abnormal soft-tissue lesions.

Gas is present throughout the alimentary canal, but under normal conditions (except in infants and very small children) one does not see gas shadows in the roentgenograms of the small intestine. In the stomach and colon, gas shadows are found frequently in roentgenograms of the abdomen. Wangensteen[1] explains the inability to see the gas shadows in the small intestine as being due to the intimacy of the admixture between the fluid and gas. Under conditions of stasis, however, the gas separates from the fluid and can be demonstrated readily.

According to Wangensteen, gas in the obstructed intestine consists of the gas produced by digestive processes, that diffusing from the blood into the intestinal lumen, and swallowed atmospheric air. Under experimental conditions, swallowed air accounts for 68 per cent of the gas shadows found

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in intestinal obstruction, the remainder of the shadows being due to the other two sources. Wangensteen says that one can demonstrate abnormal gas shadows on the roentgenograms of experimental obstructive lesions after four or five hours. It is not known how long it takes for gas shadows to become apparent in intestinal obstruction in human beings, but it is common knowledge that chemical irritants such as Diodrast cause visible gas shadows in the small intestine in a few minutes.

Abnormal gas shadows in the small intestine and colon may occur as a result of a variety of conditions, most of which the radiologist must bear in mind to make a careful correlation of the roentgenologic and clinical observations before he attempts to arrive at a provisional diagnosis. Some of the things, other than intestinal obstruction, that produce abnormal gas shadows and modify the roentgenologic signs are as follows: morphine and many other sedative drugs; peritoneal irritation from any cause, such as retroperitoneal hemorrhage due to an injury; dysentery and many bacterial and chemical irritants, and nutritional deficiencies; a ruptured graafian follicle; and general conditions, such as diabetes and sickle-cell anemia.

If the radiologist and the clinician, after study of the data, conclude that the abnormal gas shadows are due to an obstruction, the next problem to decide is where it is.

Location of Obstruction

With the exception of obstruction due to hypoproteinemia, it is not the purpose of this presentation to discuss obstructive lesions involving the stomach and duodenum.

The radiologist is frequently called on to examine patients who have had reconstructive operations performed on the stomach. Some of these patients continue to vomit, and after a barium meal, the stomach may not empty for many hours, and the radiologist and surgeon often conclude that the patient has a mechanical obstruction due to adhesions or some defect resulting from the operation.

My experience and that of Barden et al.[2] indicate that the stomach frequently empties very slowly for approximately three weeks after operation, owing to the influence of operative trauma, hypoproteinemia and possible vitamin deficiencies. After the return of such factors to normal, the stomach empties without the slightest difficulty. If such factors account for the patient's symptoms and are recognized, an unnecessary operation may be prevented.

In the study of roentgenograms of the abdomen, experience has shown that if the gas shadows are limited to the colon, and the symptoms are acute, the obstruction is likely to be found in that organ. Wangensteen,[1] Rigler and Lipschultz[3] and many others have called attention to this observation in the past. Wangensteen states that the ileocecal sphincter and valve operate in the manner of a check valve, allowing gas and fluid from the small intestine to be swept into the colon, but that regurgitation is prevented by the sphincter. In my experience, the small bowel rarely participates in a colonic obstruction unless the process is chronic or extends beyond the confines of the colon, or unless the lesion is associated with a spreading infection.

A survey examination of the abdomen may afford all the information necessary to diagnose and locate the position of a colonic obstruction. If it does not, a barium enema may be indicated to meet the requirements of the surgeon who wishes to know the nature of the pathologic process and the degree and position of the obstruction. Occasionally, it is impossible to administer a barium enema because of the patient's inability to retain it. At other times, the illness of the patient and the severe distention of the colon make one hesitate to subject the patient to the manipulation that such an examination necessitates, owing to the danger of perforation of an already overdistended and necrotic bowel.

If the abnormal gas shadows are limited to the colon, the obstruction is likely to be mechanical. I have seen only one patient who suffered from a paralytic distention of the colon produced by a spinal-cord lesion that simulated and had to be differentiated from an organic obstruction. As a rule, a paralytic ileus causes distention of the small and large intestine and offers no difficulty in the differential diagnosis.

Rigler and Lipschultz[3] describe the roentgenologic signs that they consider helpful in making a diagnosis of volvulus of the sigmoid. These signs include the increased size of the sigmoid, most of the lumen of which is found high up in the abdomen outside the pelvis and to the right of the midline, the presence of unusual amounts of fluid and the demonstration of a double point of obstruction. A barium enema may be necessary to assist in determining the diagnosis. In a patient with volvulus whom I studied, the administration of the enema was greatly facilitated by having the patient get in the knee-chest posture. A similar position is recommended in all patients who have large pelvic masses.

If the patient has a primary lesion of the colon, such as diverticulitis or an infected carcinoma, the

small intestine, in addition to the colon, is frequently distended. The use of the stethoscope assists in diagnosing or excluding a paralytic ileus, and a barium enema frequently helps to determine the nature of the pathologic process. This is especially significant when the gas shadows of the small intestine simulate the haustral markings in the colon.

A colonic lesion is apter to result in perforation than a lesion of the small intestine is, and the cecum is more vulnerable to distention than other portions of the colon. Roentgenograms are made in the erect and the lateral decubitus positions to demonstrate free gas in the peritoneal cavity. Thick shadows of the intestinal walls may be due to peritonitis. The loss of the shadow of the preperitoneal fat line or the absence of the lumbar-muscle shadows should be evaluated, because such observations may indicate peritonitis.

The clinical signs of obstruction of the small intestine are much more definite than those of colonic obstruction. The observation of varying amounts of distention of the small intestine is the only roentgenologic evidence of such a condition. Until a few years ago, the radiologist without the aid of the opaque meal was unable to demonstrate the site and nature of the obstructive lesion. Now, through the agency of a multiple-lumened tube,[4] it is possible not only to intubate the small intestine but also to determine the position and nature of the obstructive lesion and the completeness of the obstruction.

The procedure of intestinal intubation in obstruction consists in the passage of the tube down the intestine until it stops, or, if the obstruction is paralytic, until the obstruction has been overcome. In mechanical obstruction, it is possible to inject a thin suspension of barium and determine whether one is dealing with an intrinsic lesion, such as a tumor, or an extrinsic lesion, such as adhesions. If, after the barium has been injected, the opaque suspension regurgitates proximally along the barrel of the tube, one suspects an obstructive lesion. Colonic lesions are likely to cause the tube to stop progressing in the small intestine in the right iliac fossa owing to spasm of the ileum. Experience leads me to suggest that whenever the tube stops in the ileum one should administer a barium enema to diagnose or exclude a colonic lesion before making a diagnosis of an organic ileal obstruction.

One of the most frequent errors in interpretation has to do with the false impression of progression of the barium that is injected into the intestine by way of the Miller–Abbott tube. The sequence of observations may be as follows: the double-lumened tube stops in the right lower quadrant; the tip of the tube, in progressing to that point, has allowed the small intestine to telescope over the barrel of the tube; the balloon is deflated and 20 cc. of thin barium suspension is injected into the intestine; following the deflation of the balloon, the portion of the small intestine that has telescoped over the tube begins to readjust itself, thus allowing barium to escape beyond the tip of the tube even in the presence of a complete obstruction. Obviously, the barium does not progress beyond the point of complete obstruction, but the apparent progression of barium is due to the unraveling of the bowel from the tube. Evidence of pleating of the tube proximal to the balloon should always be looked for, and the patient should continue to swallow the tube until pleating has disappeared before one may be sure that the tip will not retreat up the intestine on deflation of the balloon.

The procedure of intubation has many other important functions to fulfill. Some of the purposes that have been recorded by Abbott[5] include the following: to determine the nature of the obstruction; to determine fluid and salt loss; to relieve colic and vomiting by decompression of the distended bowel; to return peristalsis by relief of overdistention; to render operations elective rather than emergency procedures; to facilitate surgical procedures (especially in multiple obstruction); and to decrease the likelihood of postoperative leaks.

Many objections have been raised to the procedure of intubation. The first is the time required to pass the tube. A physician experienced in intubation is essential. In recent months, Abbott[6] has been able to pass the tube into the duodenum much more rapidly by the assistance of the rigidity produced by a piano wire placed in the double-lumened tube. Secondly, it is not always possible to aspirate the contents from the intestine. Thirdly, the tube is sometimes found to be obstructed after it has been passed. The employment of a nurse to keep the tubes clean should obviate any such complaint. Fourthly, in colonic lesions, spasm of ileum may cause faulty obstruction of the tube. Fifthly, the procedure may fail to decompress the colon, or the small bowel in cases with multiple obstructions. Finally, there is the delay in operation due to the loss of time while intubation is being attempted.

There are many suggestions that have been and might be made to help one overcome the objections just detailed, but most of these drawbacks are easily obviated if an experienced team is developed in each hospital. When that time comes, the high mortality of intestinal obstruction will become a relic of the past.

Extent of Obstruction

Most of the incomplete obstructions are studied by either the barium meal or enema, or both; they are often unsuspected. Intestinal obstruction after operation is often incomplete, and frequently occurs as a result of inflammatory adhesions or of adhesions in addition to inflammation or chemical imbalance in the tissues. If such a patient has a partial obstruction of the small intestine the Miller–Abbott tube, if passed, will stop at the site of the obstruction. Then, if barium is injected through the tube, it will progress in a caudal direction, and the nature of the obstructing condition can often be diagnosed. Such information is extremely valuable.

If the small bowel is completely obstructed, nothing will pass through it, and if barium is injected through a Miller–Abbott tube, it will regurgitate along the barrel of the tube. The complete obstruction may be temporary, particularly in cases that have been successfully intubated and have had the eleectrolyte balance re-established.

Multiple Obstructions

The diagnosis of multiple obstructions on the plain roentgenogram of the abdomen may be suspected but is exceedingly difficult to substantiate.

The multiple obstructions that one sees may be anything imaginable, but as a rule they fall into several categories: multiple obstructions of the small bowel that are due to adhesions or metastatic cancer (frequently ovarian); those of the small and large bowel caused by tumors (Hodgkin's disease, ovarian neoplasm and so forth); those of the small intestine and colon due to the effects of irradiation; those of the small and large bowel due to an inflammatory lesion (pelvic abscess) or granulomas, such as regional ileitis; those of the colon due to a tumor and an inflammatory process elsewhere, such as diverticulitis.

If the procedure of intubation is employed, the radiologist may frequently make a diagnosis of multiple obstruction on the basis of the following observations. The Miller–Abbott tube may fail to progress beyond a certain point, and gas-distended loops of small intestine may be present beyond the tip of the Miller–Abbott tube; the tube may fail to progress beyond the left lower quadrant, with either gas-distended loops of small intestine distal to the tip of the tube or a distended large bowel, or both; and there may be an obstruction to the barium injected through the tube (with the exception of false ileal obstruction), together with an obstruction demonstrable by a barium enema.

Cause of Obstruction

The obstruction is often the final closure of a slowly developing lesion, and such a possibility should be kept in mind. Other causes of obstruction may be many things and conditions, a number of which have already been discussed.

Intussusception may produce strangulation. It is imperative to recognize this condition because a disturbance in the blood supply may be produced without much clinical evidence. This, according to Wangensteen,[1] is due to the sheathing of the gangrenous portion of the bowel by the enveloping walls of the intestine.

If barium can be given by mouth, the nature of the lesion is readily recognized by the large filling defect of the tumor or intussusception, or both, by the narrow lumen of the intestine in the vicinity of the intussusception, except when the intussusception herniates through a gastroenterostomy opening, and by a barium enema, the filling defect of the advancing intussusception in the colon being readily demonstrated. If no opaque mixture is administered, the gas in the loops of intestine proximal to the point of obstruction is readily demonstrated, but when the lesion occurs in infants and children, the observation of gas shadows may be of no assistance, since these shadows are found normally in children up to several years of age. The oblong soft-tissue shadow of the intussusception is also diagnostic.

The roentgenologic observations in strangulated obstructions from other causes are quite different from those just described. One sees tremendous distention of the small intestine when the process involves that organ. In addition, some overdistention of the large bowel may be produced by the peritoneal irritation.

There are a number of conditions that may produce roentgenologic appearances similar to those of a strangulated obstruction. In my experience, some of these have included acute pancreatitis, twisted ovarian cyst, sickle-cell anemia, diabetes, subacute drug (morphine) effects, mesenteric occlusion, ruptured graafian follicle and paralytic ileus. The findings of the clinicians and surgeons must be correlated with the roentgenologic findings before one can make a diagnosis, and, even then, errors will occur.

The question of intubation is being considered more frequently even in those cases in which strangulation is thought to be present. Although the viewpoint of clinicians and surgeons is that only those cases that can be safely delayed should be intubated, I have recently heard conservative surgeons who have had large and successful experiences state that they recommend the passage of

the tube while the patient is being prepared for operation.

Summary

In a very general way, the roentgenologic signs that might assist in the diagnosis of intestinal obstruction are considered under the following headings: the presence, location, completeness, number and cause.

Some of the experiences with intubation that are of interest to the radiologist are recorded, particularly those that may lead to errors in diagnosis.

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INDICATIONS FOR THE USE OF THE MILLER-ABBOTT TUBE*

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IN a broad sense, the indications for the use of the Miller–Abbott technic of intestinal intubation[1] may be classed as experimental, diagnostic and therapeutic.

Experimental Procedures

The experimental employment of the method, begun nine years ago at the University of Pennsylvania after two decades of preliminary trial, is worthy of mention because the special clinical applications have always developed from experimental procedures. As a result of the ability to reach any portion of the jejunum, ileum or colon with reasonable ease, the intestinal chemistry, the motor activity of the bowel wall and the responses of the intestine to drugs came under investigation in man. Aspiration of the intestinal contents from chosen regions before and after specific foods has shown the site and rate of absorption, the range of intraintestinal osmotic pressure, the tendency of the chyme to approach neutrality and the mechanisms available to the intestine for maintaining these normal relations. Such data make possible the construction of good jejunal feeding mixtures. By attaching recording instruments to the lumen of the tube leading to the balloon and by injecting radiopaque mixtures locally into the lumen of the intestine, the movements of the intestine and their effects on the behavior of the contents have been determined. The projection of these studies into the clinical field has resulted in the diagnostic procedures to be discussed later. The pharmacologic action of drugs may be investigated from the point of view of secretory, absorptive and motor phenomena in a manner impossible to accomplish in the surgically deranged digestive tract. Thus, from the experience accumulated in hundreds of experimental intubations, the therapeutic methods were developed.

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Diagnostic Procedures

From these physiologic investigations have come both the diagnostic and the therapeutic procedures dependent on the passage of long tubes. The indications for diagnostic intubation are as follows: suspected absorption defects, such as those that appear to characterize sprue and allied conditions; mild abdominal pain in which the distinction between a neurosis and a small lesion of the intestine is difficult by other means; high-grade obstruction in which it is essential to know the location and nature of the obstructing lesion; and rare conditions, such as gastrointestinal hemorrhage of unknown origin or suspected intravisceral fistulas, when the aspiration of the contents of the intestine reveals the location of a lesion not otherwise detectable.

Absorption Defects

The claim that a determination of intestinal absorptive power is of diagnostic value is as yet founded on somewhat tenuous evidence. It has long been believed that the group of diseases designated by such names as celiac disease, idiopathic steatorrhea, Gee–Thaysen disease and nontropical sprue is characterized by a loss of the specific absorbing power of the mucosa. The recent work of May and McCreary[2] has cast some doubt on this hypothesis, although the bulk of the evidence to date still supports the older point of view. If one is eventually to demonstrate the validity of

such a fault as an impediment to the patient's nutrition, one must be equipped with an accurate technic. Attempts have been made to appraise this by determining the blood levels of substances given by mouth. These measures have failed because, obviously, the blood level may be determined as much, or more, by the rate of elimination of the substance from the blood as by the rate of absorption from the intestine. One must then determine absorption by measuring the rate at which the test material leaves the intestinal lumen. Certain methods have been tried in the last few years, such as that of Groen,[3] which depends on the passage of a double-lumened intestinal tube, the distention of a terminal balloon and the retrograde filling of the intestine behind the balloon for a given period. Although this procedure suffices under most normal circumstances, the fact that conditions associated with poor absorption are usually accompanied by bizarre motility changes makes it practically impossible to prevent or to identify technical errors. A disappearance-time technic based on the qualitative testing of intestinal samples for glucose after the ingestion of a stated amount of sugar has been developed by Warren.[4] Noting the time at which the last trace of reducing substance disappears from the chyme gives a measure of the specific power of the epithelium to remove glucose. Although the experimental procedure of Zetzel and Banks[5] is excellent and could be applied as a clinical test, probably the most successful combination of simplicity with accuracy to date is the method of Nicholson.[6] This technic involves the passage of a triple-lumened tube 90 cm. beyond the pylorus, and the institution of a continuous flow of a 10 per cent glucose solution into the duodenum, with continuous aspiration of intestinal contents from a point above an obstructing balloon at the tip of the tube. A known length of intestine is thus continuously perfused with the test solution. The requirements of a good method are that it be simple enough to apply to sick patients, that a unit area of intestinal mucosa be examined, that the application of the glucose solution to the mucous membrane be quick enough to give a sharp starting time, that the residual glucose be removed from the test area at the end of the experiment rapidly enough to give a sharp stopping time, and that the mucosa be constantly supplied with a quantity of the test substance in excess of its maximum absorption capacity yet below a concentration level that would cause mucosal irritation and below a volume level that would grossly flood the intestine. By Nicholson's method, an initial acceleration of the intraduodenal flow can quickly bring the solution into contact with 90 cm. of bowel, and the final introduction of a magnesium sulfate solution will wash out within two or three minutes the glucose remaining in the test region at the end of the absorption period. Although it is true that the balloon on the end of the tube continues to creep down the intestine at an unknown rate throughout the experiment, so that one ends a half-hour period with more mucosa exposed than at the beginning, the method as a whole is far freer from technical faults than any so far described. If one is to deal accurately with patients exhibiting clinical evidence of absorption defects, a diagnostic method that will unequivocally detect such faults becomes essential.

Organic versus Functional Lesions

The second diagnostic use of intubation is the detection of minimal lesions masquerading as neuroses. This grew out of kymographic studies of intestinal movements. The balloon on the end of a long intestinal tube attached to a suitable recording instrument is a satisfactory apparatus for translating the contractions of the intestinal wall into a continuous graph on moving paper. A localized lesion in the small intestine practically always changes the character of the muscular contractions at that point, producing either local spasm or, if a long-standing partial obstruction has been present, local dilatation. The experience with this method is also too limited to permit dogmatic statements. Since the time for a record may occupy several hours, it may require several years to obtain enough normal control material. Nevertheless, the observations of Ingelfinger and Abbott[7] suggest that, in cases of midabdominal distress with functional symptoms and gastrointestinal x-ray series showing changes of doubtful significance, a balloon record will often tell, first, whether the intestinal disorder is local or general and, secondly, whether, if local, it is spastic or anatomic. From the therapeutic standpoint, this means the differentiation of a lesion possibly correctible by surgical means from one that should best be handled conservatively.

Injection of Opaque Mediums for Roentgenoscopy

A far better established diagnostic use of intubation depends on the injection of an opaque medium.[8–13] This is an important supplement to the kymographic tracing of minimal lesions just described and has drawn the special attention of Boon.[14] A disorder may readily be sufficient to stop the advance of a balloon on a tube while still permitting the advance of a liquid such as a barium suspension. Thus, if during a diagnostic intubation the tube comes to rest at any given

point in the intestine, that region should be carefully examined fluoroscopically and with films during the injection of 50 cc. of a moderately thin water-barium mixture, and again half an hour after the injection, to determine whether the barium has advanced or remained at the point at which it entered the intestine. The fluid flows in the direction of least resistance, and retrograde regurgitation is the factor of chief importance in diagnosing obstruction. Distortion of the lumen, revealed by the advance of barium, may indicate the nature of the obstructing lesion. It should always be borne in mind that local spasm incident to contiguous disease, such as mesenteric lymphadenitis, may simulate a local lesion of the intestinal wall.

A far easier and probably more useful roentgen-ray diagnosis, however, is made by essentially the same procedure, carried out in the presence of complete obstruction. It is obviously unjustifiable to give a barium meal in the presence of a completely blocked intestine. In cases intubated for the relief of distention, it is proper, however, to inject barium down the tube for the detection of the exact site of the obstruction and for the determination of the nature of the lesion. If the block is complete, the injected barium is regurgitated proximally. It is becoming constantly more apparent, however, that decompression of such blockage usually renders the obstruction partial by the relief of local irritability, and improved nutrition aids by reducing the local edema. A little of the barium may therefore be massaged ahead of the tube, and the nature and the extent of the lesion be revealed. It is my opinion that this procedure should be routinely carried out preoperatively in such patients.

Location of Rare Lesions

Finally, there are those rare conditions whose diagnosis depends on the ingenuity of the physician almost as much as on his previous experience. When a patient bleeds by bowel in the presence of a negative barium enema and of an apparently normal stomach and duodenal cap, the chance of a benign tumor of the small intestine or a Meckel's diverticulum or even a bleeding polyp must be considered. If a tube is advanced continuously down the intestine until blood appears in the drainage, the balloon serves as a signboard to the surgeon, indicating the position of the lesion and making unnecessary the tedious palpation of the bowel that so often results in a stormy convalescence. In similar fashion, a variety of odd clinical circumstances have been clarified by the ability to reach points in the small intestine that were formerly not accessible.

Therapeutic Procedures

The therapeutic uses of the long intestinal tube must be considered. Some of these are so specifically surgical as to be out of the province of an internist. One may, however, suggest five chief uses: the prophylaxis against expected intestinal obstruction under unknown or complicated circumstances; the diversion of the intestinal current at a point proximal to the site of an anastomosis, as in surgery of the colon; the decompression of a distended and inactive intestine; the decompression of a distended and obstructed intestine; and the maintenance of nutrition and intestinal function during the course of local peritonitis with obstruction.

Prophylaxis Against Intestinal Obstruction

Under the head of prophylaxis against intestinal obstruction are included conditions that need only be mentioned, since they are frequently individual problems in themselves. Suffice it to say that in late cases of regional ileitis or in attempts to close traumatic fistulas a surgeon may sometimes anticipate a stormy convalescence before he begins the operation. A tube in the small intestine passed the night before may give him control of a postoperative condition that might otherwise determine a fatal outcome. Similarly, after a total colectomy, edema of the oversewn transverse mesocolon may compress the third part of the duodenum. Death has resulted when this has occurred in conjunction with delayed function of the ileostomy, since gastroduodenal drainage was useless. I now preoperatively intubate all colectomy patients.

Protection of Intestinal Anastomoses and Colon Surgery

Protection of incised areas of bowel during the postoperative period can be accomplished if a tube lies proximal to the suture line. In the performance of ileocolostomies and one-stage resections of the right side of the colon, much has been said of the importance of preventing colonic distention during the first postoperative days. Probably a more significant, though less obvious, cause of leakage is distention of the ileum proximal to the anastomosis. The patient may show no gross tympanitis because the volume contained within the ileum is relatively small, but for physical reasons the pressures developed are high compared with those in the colon. Thus, a little gas and fluid proximal to an ileocolostomy is far likelier to cause a leak than a large accumulation distal to the suture line.[15] Although decompression of the whole colon, allowing rectosigmoid resection without cecostomy, has occasionally been possible,

the protection of anastomoses is much more valuable.

This is the chief function of the intubation of the small intestine in one-stage resections of right-sided colonic lesions, and although the improvement in mortality statistics — such as 29 cases with 1 death reported by Ravdin and Abbott[16] and 53 cases with 2 deaths recorded by Whipple and Nelson[17] — cannot be attributed solely to the use of the tube, I consider it fair to say that it has been an important contributing factor.

Decompression of Paralytic Ileus

Decompression of simple paralytic ileus, as distinguished from that associated with clinical peritonitis, is worth discussion. I am in complete agreement with Wangensteen[18] that the gas which distends these patients is very largely swallowed air, that the use of the Wangensteen tube for suction often prevents the occurrence of the condition if aspiration is started early enough, and that in mild cases it clears up the condition when once it is established. I disagree with him about the effectiveness of the Wangensteen technic in those cases seen after paralytic ileus has become extreme, with wide distention of the stomach, small intestine and colon, absent audible peristalsis, and the conditions attendant on marked abdominal distention. Whether these cases develop following intraperitoneal procedures or reflexly following injuries or operations on other parts of the body, a tube in the small intestine will progress from region to region, restoring peristaltic activity until the balloon is expelled by rectum. Because of the difficulty in determining when a case is severe enough to warrant intubation of the small intestine rather than the use of the Wangensteen tube, Pitressin, Prostigmine, choline derivatives, irritant enemas, stupes or rectal tubes, it is difficult to appraise the value of this procedure statistically.

Suffice it to say that 29 cases with 6 deaths have been reported by Penberthy, Johnston and Noer,[19] 9 cases with no deaths by Glenn,[20] and 55 cases with 5 deaths by Whipple and Nelson.[17] To these, I add my own experience of 16 cases with 3 deaths.

Decompression of Obstructed Intestine

Decompression of the distention resulting from a mechanically obstructed intestine can be carried out by these tubes with a reduction in the mortality rate. Especially in this group, a careful selection of cases is essential. Suspected gangrene of the intestine, except in certain cases of mesenteric thrombosis, is always a definite contraindication to delaying surgery, and anyone undertaking to use intubation clinically must exert himself to perfect his ability to identify a strangulation. This diagnosis was of somewhat academic interest in the days when all cases of obstruction were immediately operated on, since the laparotomy promptly revealed the answer. Now that operation may be delayed for days, the patient's survival may depend on whether an impaired blood supply to the involved part is recognized. It is essential to remember that, when one is presented with a case of obstruction, the choice is not between intubation and operation, but between intubation and operation at that particular moment, and on each subsequent visit to the patient's bedside one must re-evaluate the indications for radical or conservative measures. Granting that strangulation is not present, it must be borne in mind that one is intubating for the relief of distention, a condition that, in the absence of strangulation, determines the high mortality in mechanical obstruction. If, therefore, distention does not exist, intubation is not indicated unless some other factor in the case justifies the procedure.

Patients who promptly present colic and hyperperistalsis, and in particular those with conditions giving circumstantial support to the diagnosis of simple obstruction, do not require the passage of a tube. It is in the late cases with marked distention, borborygmi, fecal vomiting, dehydration, electrolyte imbalance and shock — those patients who present on admission the worst surgical risk — that a method of improving the patient's condition before he is operated on becomes most imperative. This is the function of intubation. The tip of the tube advances down the intestine in spite of fecal vomiting until the obstruction is reached. The situation is then under control to such an extent that operation may be delayed for a day or a week or whatever interval is necessary to bring the patient into a sound state of nutritional balance. If the obstruction has not spontaneously subsided, and in certain cases even if this has happened, operation should be carried out.

Justification for this statement lies in the reports of 5 obstruction deaths out of 54 patients by Johnston, Penberthy, Noer and Kenning,[21] of 5 deaths out of 96 patients by Whipple and Nelson,[17] of 3 deaths out of 27 patients by Glenn[20] and of 5 deaths out of 74 patients in my own experience.

Relief of Peritonitis with Obstruction

Finally, intubation enables one to control intestinal function and maintain nutrition in the patient with local peritonitis. Death from rupture of a gangrenous appendix or the spread of a pelvic peritonitis, even after the best surgery, is frequently the result of a combination of two main

factors: the peritoneal infection, and the functional blockage of the intestine with uncontrollable distention and the starvation that results. When one controls the obstruction, distention and starvation, the patient, particularly with the aid of sulfonamide drugs, can frequently master his infection. Such obstruction is as a rule self-limited and subsides in from one to three weeks under controlled circumstances. If it is necessary to keep an intestinal tube in place for that long, the question must be asked whether the risk of mucosal erosion by the tube at the larynx or farther down the intestine is greater or less than the danger of the condition for which the patient is intubated.

Although the technic of intravenous alimentation is a godsend to all of us, it cannot be denied that the number of calories per twenty-four hours that can be given by vein is hopelessly inadequate for long-time maintenance, and it is frequently unbalanced from the nutritional standpoint. The opportunity given by the presence of the tube to feed a patient 1200 to 1500 calories of low-residue food daily by mouth while the residue is drawn off by the tube is therefore of great advantage. Frequently, after ten days or a fortnight or even longer of complete obstruction, the patient's bowels

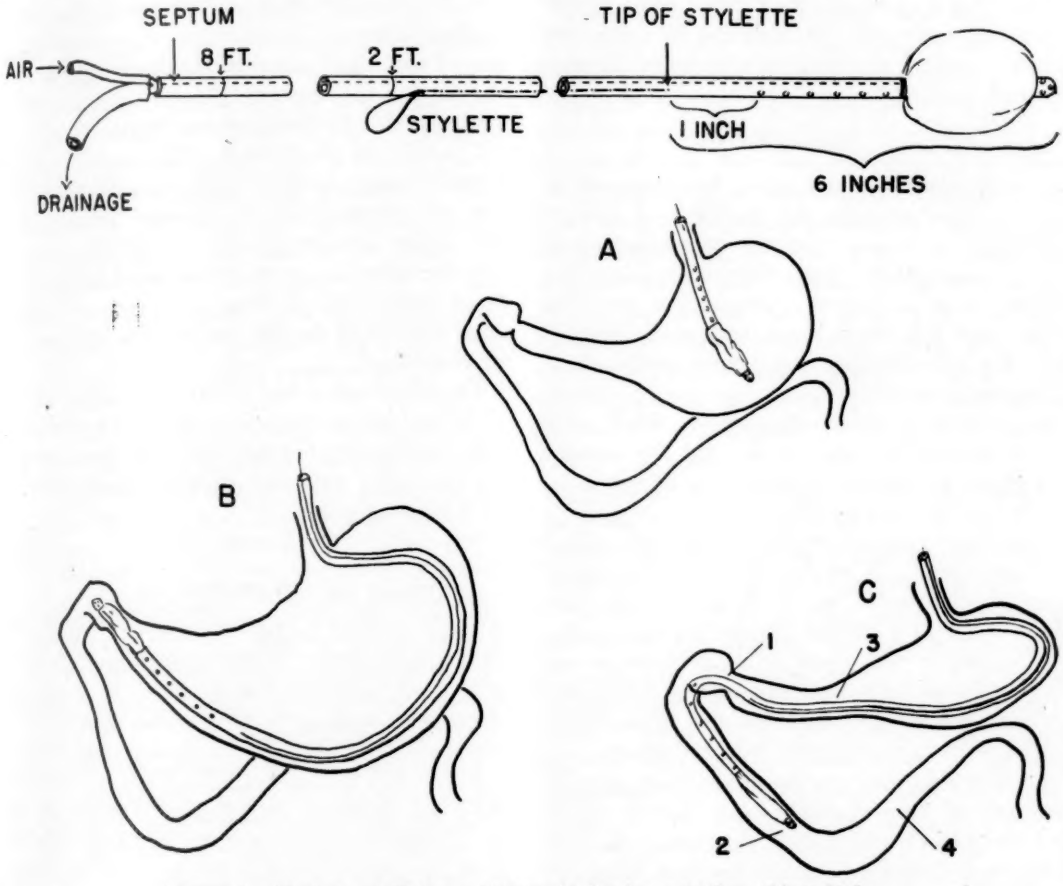

FIGURE 1. *Technic of Using a Stylette in Passing a Miller–Abbott Tube.*

Pierce the wall of the aspirating lumen of the tube at the 50-cm. (2-ft.) mark with a length of 0.4-mm. (0.016-in.) diameter, specially straightened, stainless-steel vom Hofe leader wire, and advance the tip of the wire to a point 2.5 cm. (1 in.) above the most proximal aspirating hole in the tube. Bend a loop in the other end of the wire so that the tip cannot advance distally. The terminal 15 cm. (6 in.) of the Miller–Abbott tube will then contain no wire. Pass the tube in the usual manner to the position shown in A. *With a temporary adhesive-tape patch over the point at which the stylette pierces the tube wall, inject 300 cc. of air into the stomach and advance the tube until it lies along the greater curvature, as in* B. *Hold the tube at the patient's nose to prevent its slipping out, and apply suction to remove the air. The stomach contracts, as in* C, *squeezing the tip of the tube ahead from position* 1 *to* 2. *The tip of the stylette then lies at* 1. *The antral spasm, with concentric contraction distal to* 3, *prevents the terminal 15 cm. (6 in.) from coiling, and the stylette prevents coiling of the tube proximal to the antrum, where the stomach is more flaccid. Draw back the tip of the stylette from* 1 *to* 3, *and by gentle, steady pressure on the tube at the nose advance the tip from* 2 *to* 4. *Withdraw the stylette, patch the hole through which it was introduced with thin rubber and, having inflated the balloon with 20 cc. of air, proceed as usual.*

begin to move, and intestinal function resumes its normal activity and maintains it after the removal of the tube.

The justification for the use of intubation in cases of this type is based on the report of 24 cases with only 6 deaths by Penberthy and his coworkers,[19] of 8 cases with only 1 death by Glenn,[20] of 92 cases with only 20 deaths by Whipple and Nelson[17] and of my own experience of 27 cases with only 6 deaths.

Technic

In addition to the indications for intestinal intubation, there remains the question of how to accomplish it. I know of general practitioners who are successfully and judiciously using this method; it is as a rule reserved not only for the hospital but for a physician on the hospital staff who is thoroughly experienced in the many difficulties and minor variations of the technic. If used without strict attention to detail, it is capable of causing great harm, and the interval throughout which one is entitled to persist in attempts to pass a tube rather than in carrying out surgical intervention must be determined in each case individually. This should not be determined by any set number of hours. It has been my own rule, so long as a patient is getting better rather than worse, to wait, irrespective of whether the tube is advancing well or not, but if the patient is getting worse rather than better, I advise operation regardless of what the tube is doing.

Recent developments in the technic have brought the speed of intubation far more directly under the control of the intubator. Following the basic principles laid down by Rousselot and Bauman[22] in 1933 for the use of a stylette in passing duodenal tubes, it has been possible to modify a stylette for use in long intestinal tubes (Fig. 1). With this instrument, one can usually bring the tip of the tube to the duodenojejunal junction within ten to twenty minutes. In a group of 18 severely ill patients with both obstructing peritonitis and mechanical obstruction, failure to accomplish this occurred in only 2. In the remaining 16, it was possible to pass the tip promptly to the distal duodenum, although previous experience had led me to anticipate that many hours would be required to do so. Since a stylette in a gastrointestinal tube is a potentially dangerous instrument, and since it is often far better for the patient if his stomach is thoroughly decompressed before the tip advances to the duodenum, the use of this procedure should not be routine. In the desperately ill patient, however, the ability to decompress the small intestine without delay may be crucial in determining the outcome.

Summary

Intubation of the small intestine was developed as a technic for studying the physiology of the digestive organs, and from the results have grown diagnostic and therapeutic procedures.

The diagnostic indications for intubation are aimed at the detection of disorders of absorption, minimal lesions ill defined by roentgenoscopy, advanced intestinal obstructions forbidding the use of opaque meals, and rare gastrointestinal disorders unidentifiable by standard methods of study.

The therapeutic indications for intubation are to forestall expected ileus incident to abdominal disease, to safeguard against leakage through intestinal suture lines, to abolish paralytic ileus, to control distention in mechanical obstructions, and to make possible the feeding of patients with peritonitis over long periods while the inflammation is subsiding.

The development of a stylette suitable for use in Miller–Abbott tubes has frequently made possible the prompt passage of the tip into the duodenum under the most adverse circumstances.

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THE DIAGNOSIS AND MANAGEMENT OF ACUTE OBSTRUCTION OF THE SMALL INTESTINE*

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BOSTON

IF one excludes strangulated external hernias, the diagnosis of which is easy and the treatment obvious, the diagnosis and management of acute mechanical obstruction of the small intestine remain major problems of clinical surgery. Few questions have stimulated as much experimental work by excellent investigators as, "What is the cause of death in acute small-bowel obstruction?" Although this question has not been completely answered, from the results of these investigations have come certain basic facts that, when considered in the light of clinical experience, suggest a rational scheme for the management of these cases.

The development of such a plan presupposes an understanding of the significant clinical manifestations of the disease and of certain correlated experimental findings, as well as the ability to make a diagnosis.

CLINICAL TYPES OF OBSTRUCTION

The clinical course of complete obstruction of the small bowel varies greatly, depending on the level of the obstruction and whether or not the obstruction involves the blood supply of the obstructed loop. An understanding of this problem is essential to proper management.

Simple high obstruction‡ (duodenal or high jejunal) is usually acute in onset. Vomiting of the juices of the upper gastrointestinal tract is early and profuse. The resulting dehydration is associated with rapid loss of electrolytes, of which the chlorides are the most important. A diminished urinary output and a rise in the nonprotein nitrogen of the blood follow, and there may or may not be an associated alkalosis. Such an obstruction runs a rapid course, in which distention is usually not a major factor; death in most cases results from a loss of essential body fluids. As a rule, early replacement of chloride loss by the parenteral use of physiologic saline solution or 5 per cent glucose in saline postpones or prevents death.

Low ileal obstruction is more complicated. The contents of the obstructed loop cannot be emptied by vomiting. There is some absorption of the electrolytes above the obstructing point, so that the changes in the chemical constituents of the blood are neither rapid nor profound. The accumulation of fluid and gas in the obstructed segment results in increasing distention of the bowel. With the rise in intraluminal pressure, there is slowing of the circulation in the distended segment, congestion and edema of the bowel and, later, injury to the mucosa. It is probable that absorption into circulation ceases when the intraluminal pressure is sufficiently high. A replacement of chlorides in these patients does not materially influence the usual course of the obstruction. Some other factor or factors are responsible for death in these cases.

Strangulation obstruction, due to constriction of the mesentery of a loop of bowel and resulting in interference with its blood supply, regardless of the level of bowel involved, is a rapidly fatal condition, unless the constricting band is divided before irreversible changes take place in the bowel, or unless the devitalized bowel is removed from the peritoneal cavity. Death may occur from shock before development of the peritonitis that follows permeation of the devitalized bowel by intestinal organisms.

DIAGNOSIS

The old teaching, "Any patient with an abdominal scar and an attack of abdominal pain has an acute small-bowel obstruction until proved otherwise," should not be allowed to jeopardize those patients who have never been operated on but in whom such a condition may arise. Approximately 20 per cent of the patients seen at the Massachusetts General Hospital have had no previous operation.

The five important diagnostic criteria are as follows:

Pain, sudden in onset, severe and colicky in nature, usually at or above the level of the umbilicus but at times in the lower abdomen or generalized, is the presenting symptom in nearly every case of acute small-bowel obstruction. This pain may be constant if strangulation is present or in the later stages of a simple obstruction. It may even disappear late in the course of the disease.

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‡Simple obstruction means occlusion of the lumen of the bowel without interference with its blood supply.

Vomiting follows the pain. It may be small in amount or, in the later stages, copious and fecal in character. The so-called "fecal vomiting" frequently associated with intestinal obstruction is usually a late symptom and of no great help in an early diagnosis.

Hyperactive, tinkling, high-pitched peristalsis, audible over the abdomen, is often present, and although it is not necessary for a diagnosis, its presence is highly suggestive.

X-ray evidence of one or more distended loops of small bowel, as shown by a scout film of the abdomen, is the most useful single objective finding in a patient with small-bowel obstruction. The x-ray film should be taken after the stomach has been emptied of its fluid and gas content, and before an enema has been given. This avoids the confusing factors of gas and fluid in the stomach and the possible error due to the instillation of gas and fluid into the colon and its subsequent incomplete evacuation. I have observed dilatation of coils three hours after the onset of pain. On the other hand, I have seen two proved cases of obstruction during the past year (one with strangulation and the other a simple obstruction) in which there were no abnormal gas shadows. The inexperienced should be warned that the interpretation of existing shadows may be difficult. The small bowel may be so dilated that an x-ray diagnosis of large-bowel obstruction is made. In some cases in which the interpretation of gas shadows has been difficult, positive identification of the colon by injection of a small amount of barium by rectum has proved most helpful, and from this maneuver there have been no ill effects. In other cases, a repeat film after six hours may show a change in size or distribution of the gas which will facilitate a more accurate diagnosis.

Constipation. Characteristically, there is cessation of the passage of gas or feces by rectum following the initial pain. Small amounts of gas or feces may be passed, however, since in many cases the bowel below the obstruction empties itself of its contents. Moreover, the obstruction may at first be incomplete, and small quantities of gas may pass into the lower segment. Abdominal cramps with hyperactive peristalsis and little or no gas passing by rectum are strongly suggestive of an obstruction at some point in the intestinal tract.

Strangulation obstruction. When the diagnosis of small-bowel obstruction has been suspected or made, a more detailed interpretation of whether the obstruction does or does not interfere with the blood supply to the obstructed loop is of the greatest advantage. In my experience, such a differentiation can readily be made in the later stages of the process, but only rarely in the early hours of the disease. Gangrene may develop very rapidly, as in a patient operated on six hours after the onset of symptoms and requiring resection of a gangrenous segment of bowel.

The pain in strangulation obstruction is usually steady, with or without associated spasms of colic. I have treated several patients with strangulation obstruction who complained of pain in the mid-back, a symptom not commonly encountered in the absence of interference with the mesenteric structures. Although tenderness may be present over the constricting point in simple obstruction, any increase or spread of this tenderness should be interpreted as evidence of strangulation, particularly if the tenderness is associated with muscle spasm. The leukocytic count is too uncertain to be of value. An abdominal tap is easily and safely done, and may prove useful in doubtful cases. A small-gauge hypodermic needle of adequate length or a 20-gauge or 22-gauge lumbar-puncture needle is used. The finding of blood-tinged fluid warrants a diagnosis of strangulation obstruction. A negative tap does not exclude it, however.

The diagnosis of strangulation obstruction is difficult to make early and costly to make late; it is therefore safer to operate promptly on all patients seen in the first twenty-four hours of their attack, since during this period, gangrene—whether incipient or actual—can usually be cured.

Causes of Death

Experimental. Any attempt to review the experiences of the laboratories in the effort to determine the presence or absence of a toxic factor in death from simple obstruction of the small bowel would only confuse the clinician. However, from these laboratories have come many valuable facts that are accepted by most workers and are essential to an understanding of the treatment of these conditions. They may be briefly summarized as follows:

In simple high intestinal obstruction, death comes as the result of dehydration and chemical imbalance from loss of the juices of the upper gastrointestinal tract, whose replacement with physiologic saline solution prevents or greatly postpones a fatal outcome.

In simple low obstruction, other factors are involved, since replacement therapy does not greatly alter the length of life of such animals.

Distention is a constant and essential factor in the fatal cases without gangrene.

Distention is due primarily to gas — most of which is swallowed air — and intestinal fluid. Associated with or secondary to distention are marked increase in the bacterial count of the bowel content, diminution of the circulating plasma and blood volume, and slowing of the circulation in the bowel wall, with consequent interference with absorption and, later, injury to the mucosa.

Strangulation obstruction is associated with rapid and marked diminution of the circulating plasma and blood volume, and early injury to the bowel wall, with increased permeability to bacteria and ensuing peritonitis. Death follows in most cases from the effects of strangulation rather than as a result of the associated obstruction.

The dictum of the laboratories, therefore, is clear: lost chlorides should be replaced; distention should be relieved or prevented and in strangulation obstruction, lost plasma and blood should be replaced at once, the strangulating mechanism should be relieved, and the necrotic bowel should be removed.

Clinical. Whatever the lethal factors in experimentally produced obstruction may be, the fact remains that 29 patients dying from acute small-bowel obstruction at the Massachusetts General Hospital in the last seventeen years may be readily grouped under four different headings (Table 1).

TABLE 1. *Causes of Death in Acute Small-Bowel Obstruction at the Massachusetts General Hospital, 1924–1941.*

CAUSE OF DEATH	NO. OF CASES
Peritonitis	17
Pulmonary collapse or pneumonia	5
Shock	4
Miscellaneous	3
Total	29

All but 4 of these patients were operated on. In 3 (all with strangulation), the diagnosis was made at autopsy, and the fourth patient died of aspiration of vomitus during passage of a stomach tube. Eighteen of the 29 deaths were secondary to strangulation obstruction.

Not satisfactorily explained, either in the laboratory or from the hospital records, is the sudden collapse, usually fatal, that is occasionally seen following operation and the sudden release of a distended obstructed bowel either into the lower segment or externally.[1] Whatever its cause, it is a clinical entity that cannot be overlooked in the management of the distended bowel.

In a recent analytical study of 136 cases at the Massachusetts General Hospital,[2] certain pertinent clinical facts were re-emphasized.

Following the teaching that early diagnosis and operation are indicated on patients who are good risks, and relief of distention by simple blind ileostomy without exploration on those who are poor risks, the mortality in simple obstruction over a ten-year period has been reduced from 31 to 12 per cent.*

In 42 patients seen and operated on within twenty-four hours after the onset of symptoms, there were no deaths; whereas in 17 patients entering within this period whose operations were delayed into the second twenty-four hours,† 5 (29 per cent) died.

Fourteen (64 per cent) of 22 patients sixty years of age or older, and 6 (75 per cent) of 8 patients seventy years of age or more, died following operation.

Careful insertion of a No. 16 or 18 Fr. catheter into a loop of distended bowel, using the Witzel or a comparable technic, without exploration of the abdomen, was a lifesaving procedure in late cases with marked distention. On the other hand, this same procedure in association with any attempt at exploration or manipulation of the bowel ended fatally, usually from peritonitis, in most patients so treated.

Thus, from a study of these records of patients, living and dead, it is apparent that prompt hospitalization, early diagnosis and early operation give good results, and that late diagnosis and early operation on a highly infected, distended or gangrenous bowel give poor results.

TREATMENT

From a knowledge of the foregoing experimental and clinical facts, the ideal treatment for acute intestinal obstruction can be readily formulated. In addition to prompt diagnosis and early operation (two or three hours after hospital admission) on all patients seen within twenty-four hours of the onset of symptoms, immediate operation on any patient with strangulation obstruction should be performed. Substitution therapy, consisting in glucose in physiologic saline solution sufficient to restore lost electrolytes, should be instituted promptly. Prompt transfusion with plasma or whole blood into patients with marked distention or strangulation obstruction is indicated.

*The increasing use of the inlying stomach tube and Wangensteen suction undoubtedly contributed greatly to these results.

†Delay in these patients was due to inability to make a diagnosis of obstruction or because the condition was not considered sufficiently serious to warrant immediate operation.

Evacuation of the obstructed segment, to release distention, should be carried out before one attempts to relieve the obstructing factor in patients, seen twenty-four hours or more after the onset of symptoms, who have distended bowel on physical or x-ray examination.

The pioneer work of Wangensteen[3] on suction drainage of the upper gastrointestinal tract, given a new impetus by Miller and Abbott[4] and Johnson and his associates[5] by the demonstration of a practical method of draining the lower segments of the small bowel, has overcome the one difficulty — the relief of distended bowel in the patient with a simple but otherwise fatal obstructing lesion — in the application of the above principles to clinical medicine.

In November, 1939, the Committee on Small-Bowel Obstruction was organized at the Massachusetts General Hospital for the supervision and study of all such cases entering the hospital. This committee consisted of Dr. Richard Warren, the current residents on the two surgical services and myself. The actual treatment of the patients was carried out by the men on duty, as in the past.

Based on the principles outlined above, the following recommendations (previously expressed by two of the committee[2]) were given a trial:

Prompt replacement therapy is instituted for all cases and is continued according to the degree of chemical or fluid imbalance.

A double-lumened Miller–Abbott tube is passed immediately, and the stomach emptied of its fluid and gas contents. The patient is sent to the X-ray Department for a scout film, and an attempt is made to pass the tube through the pylorus at that time if evidence of small-bowel obstruction is present. If operation is to be done, suction is applied and maintained throughout the procedure to prevent regurgitation and possible inhalation of intestinal contents.

Operation is performed two or three hours after admission on all patients seen within the first twenty-four hours* of the onset of symptoms, and on all patients with a diagnosis of strangulation.

Operation within six to eight hours is carried out in most cases seen in the second twenty-four hours after the onset of symptoms, except in patients sixty years of age or older and in those with marked distention.

Nonsurgical drainage is instituted for all patients sixty years of age or over who enter the hospital twenty-four hours or more after the onset of symptoms, and in all patients whose symptoms are forty-eight hours† or more in duration.

If intubation is unsuccessful, if the patient is not relieved by continued aspiration of the stomach over a period of six to eight hours, if the degree of distention clinically or by x-ray study is not excessive, and if the patient is in good surgical condition, immediate laparotomy under general or spinal anesthesia should be performed.

If intubation is unsuccessful and if the age or condition of the patient or the degree of distention makes release of the obstruction hazardous, a blind enterostomy is done under local anesthesia. A catheter (No. 16 to 18 Fr.) is placed into a distended loop of bowel with aseptic technic and without exploration. If bloody fluid is found, the loop of gangrenous bowel must be found and delivered from the abdomen, regardless of the condition of the patient.

In good-risk patients, an operation of election should be done in those cases that have been successfully decompressed, regardless of whether the mechanism of obstruction has been outlined by the instillation of barium through the tube.

Results of Treatment

During the last eighteen months, 29 patients have been seen and followed by this committee. A correct diagnosis of strangulation was made once. It was not made but was present in 3 cases. Seven cases are excluded from this study because they were secondary to a carcinomatosis, pelvic infection or other conditions in which obstruction was only a contributory factor. Twenty-two cases of the type under discussion were seen. The number is too small to permit more than a report of progress, but these cases have been carefully studied, followed and reviewed at frequent committee conferences, and have been helpful in guiding us in the continued care of these patients.

Diagnosis. A correct diagnosis was made in 20 cases. Improper interpretation of scout films resulted in an incorrect diagnosis of large-bowel obstruction in 1 case, and 1 patient died of an undiagnosed strangulation.

Positive x-ray findings were present in all but 2 cases. The films in one of the positive cases were misinterpreted, and in another, an incorrect

*The time limits set are of necessity somewhat elastic, but have proved to be of greatest help in deciding for or against early operation. It is only within the first twenty-four hours of the attack that most patients with strangulation can be saved, and that practically all patients with a simple obstruction can be safely operated on.

†Theoretically, all patients with simple obstruction not moribund on admission and without some serious constitutional disease should survive if properly treated. This is not so with strangulation obstruction unless the patient is operated on before irreversible changes have taken place (usually within twenty-four hours). After twenty-four hours have elapsed, 70 per cent of our patients with strangulation have required resection, with a mortality of 70 per cent. It is therefore safer to accept the hazard of strangulation than the risk of operation on the distended patient with a simple obstruction.

interpretation was made during the night by the Surgical Service and the roentgenologist on call, to be corrected the following morning by one of the senior members of the X-ray Department. There is no question that the correct interpretation of these films is at times very difficult, but it is equally striking that without the use of a scout film the diagnosis could not and would not have been made in some of these patients. The 2 cases with no evidence of abnormal gas shadows are also of importance in indicating that negative x-ray study must not be construed to exclude a diagnosis of acute obstruction.

Miller-Abbott tube. A Miller–Abbott tube was passed in all patients but 2 infants with intussusception. As soon as the diagnosis was made, an effort was made with the fluoroscope to place the tube at or to pass it through the pylorus. In 10 of 20 cases, it did not pass the pylorus, owing in 3 cases to early operation, in 1 to spontaneous release of the obstruction, and in the 6 others to technical inability to get it through.* It should be emphasized, however, that all these patients are benefited by the continuous aspiration of the stomach. I should probably be embarrassed to admit such a high percentage of failures, if I did not know that failure resulted only after a sincere effort by men of experience equal to that which I believe to be available in most well-organized hospitals. Because of this, 2 blind ileostomies were carried out successfully, one before and one after operation. Because of the difficulty of intubation, I believe that much valuable time may, but need not, be lost in prolonged unsuccessful attempts to obtain passage of the tube. This is particularly true in the earlier stages of the obstruction, when a corrective operative procedure can frequently be safely carried out.

Mortality. All but 1 of these patients were operated on, the exception being a seventy-two-year-old woman who responded well to suction drainage and whose age made elective operation inadvisable. There were no deaths from simple obstruction in this group of patients, although in 2 patients, both over sixty years of age, a note is made in the record that decompression by means of the Miller–Abbott tube (extending in one case for five days and in the other for eleven days) was lifesaving. From a review of these cases, it is quite likely that had early operation been instituted, as the custom has been in recent years, death would probably have resulted in each case. Interestingly enough, were this so, the mortality in this small group would be the same as that for simple obstruction in the last decade at the Massachusetts General Hospital.

There were 4 patients with strangulation obstruction, 2 of whom died. One of them was a man of sixty-seven who entered the hospital acutely ill twenty-four hours after the onset of symptoms, on whom operation under local anesthesia was done in an attempt to save his life after all other efforts to improve his condition had failed. The second death was in an eighty-year-old woman who entered the hospital only seven hours after the onset of symptoms, who was followed for twenty-three hours after admission, whose x-ray films showed no abnormal gas shadows, and who was finally operated on without a positive diagnosis, a strangulation obstruction requiring resection being found.

*In recent months, the use of a fine stylette, as suggested by Abbott[6] has greatly facilitated this step in cases in which difficulty has been encountered.

Summary

The important experimental and clinical evidence affecting the treatment of acute obstruction of the small intestine is summarized.

A working plan for the management of these cases is presented, and the results in a small group of 22 cases are reviewed. There were 18 cases of simple obstruction, with no deaths, and 4 cases of strangulation obstruction, with 2 deaths.

The value of early hospitalization and early operation in patients seen within twenty-four hours of the onset of symptoms is again emphasized.

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THE TREATMENT OF TETANUS*

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THE treatment and nursing care of a patient severely ill with tetanus present a difficult medical problem. When we were faced with such an emergency, we turned to the literature for guidance but were disappointed by finding no adequate description of the clinical course of such a patient. Because of this apparent lack, we offer the following case report in detail, hoping that it may lend courage and help to those engaged in caring for patients with severe tetanus.

CASE REPORT

B. M. (M. G. H. 258145), a 10-year-old boy, was running on the ocean beach on June 29, 1940, when he struck the side of his left foot at the base of the small toe. At first, this was thought to be a stone bruise, but the next day it was found that a splinter had penetrated the skin. The splinter was removed, and iodine applied; 3 days later he visited his family doctor, who advised hot boric soaks. By July 3, the area showed signs of inflammation and yielded a small amount of pus. The patient occasionally soaked the foot as advised but was active in play until July 5, when he did not feel well and complained of a backache at evening and passed a restless night. On the morning of July 6, he complained of headache, backache and a stiff neck. When seen at home at 11 a.m., he was in typical tetanus, with opisthotonos and risus sardonicus, and he had severe muscular spasms when touched. He was given phenobarbital and ⅙ gr. morphine, and transferred to the Massachusetts General Hospital at 1 p.m.

Sedation was started with 3 gr. sodium luminal subcutaneously, and at 2 p.m., under ether anesthesia, the area about the base of the little toe was excised and two black splinters of wood were removed. Cultures of these splinters, the longest 1 cm., yielded *Clostridium tetani.* An intracutaneous test with horse serum was negative. Although the patient's older brother was a known asthmatic, it was decided to give horse-serum tetanus antitoxin. He received 20,000 units of antitoxin about the wound, 20,000 units encircled about the left midthigh and 20,000 units in an intravenous injection of 5 per cent glucose in physiologic saline solution. At 4 p.m., another 40,000 units of antitoxin was added to the fluid being given intravenously. A lumbar puncture was performed, and the fluid removed was replaced by an equal amount of fluid containing 10,000 units of antitoxin.

The patient remained under fair sedation, with a total dose of 12 gr. of sodium luminal subcutaneously and ½ gr. morphine in the first 12 hours. The back was arched, and he had intermittent, severe muscular spasms throughout the night.

A lumbar puncture at 10 a.m. on July 7 showed an initial spinal-fluid pressure equivalent to 400 mm. of water, which was reduced to 200 mm. by removing an opalescent fluid containing 1350 polymorphonuclear leukocytes per cubic millimeter; 50,000 units of antitoxin was given intravenously at 10 a.m., another 20,000 units at noon, and another 20,000 units at 3 p.m. The first severe convulsion came at 5 p.m.; fortunately, the resident physician was in the room and administered ether anesthesia and adrenalin. Several convulsions followed, and at 10 p.m. another lumbar puncture was done; again the initial pressure was 400 mm. and was reduced to 100 mm. by drainage of spinal fluid. An additional 10,000 units of antitoxin was given intravenously at 11 p.m. During the night, there were convulsions every 15 to 30 minutes, and the nurse administered ether and frequently aspirated the nose and the throat through the airway tube, which had been inserted at the time of the first convulsion. In this 24-hour period, the patient received 27 gr. of sodium luminal subcutaneously and also 7½ gr. of Sodium Amytal. Dressings of the foot wound with powdered sulfapyridine were done twice daily.

A severe convulsion at 7 a.m. on July 8 led to intense cyanosis, and oxygen, flowing at a rate of 4 liters per minute, was accordingly made available through a tube passed into the ether cone. At 8 a.m., another lumbar puncture was done, and the pressure was reduced from 350 to 100 mm. This definitely relieved the patient. Because of the sustained fever, the nurses — two special nurses worked together — applied to the trunk and extremities towels wrung out in cold alcohol; usually, 20 minutes of such application was sufficient to cause a drop of at least 2°F. in temperature. Throughout the day, his condition was poor, the respiratory rate and pulse increasing. At 4 p.m., convulsions began after an attempt to catheterize a very full bladder. The cyanosis and la-

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bored respiration were helped by intravenous adrenalin, which was stopped when the pulse became rapid and feeble. At 9 p.m., a severe convulsion caused respiration to cease for 3 minutes. A lumbar puncture was done at 10 p.m., and the spinal-fluid pressure was lowered from 300 to 78 mm., with definite relief. Ice packs were applied to the head at this time, and the patient seemed improved by these measures. In these 24 hours, he received 18 gr. of sodium luminal subcutaneously — much of it at the discretion of the nurses.

On July 9, convulsions of moderate intensity continued, about two in each hour, until 9 a.m., when another lumbar puncture was performed with considerable difficulty, owing to the marked degree of opisthotonos. The initial pressure was found to be lower — 250 mm. After drainage, with reduction of the pressure to 100 mm., the patient seemed easier, but respirations were rapid and considerable mucus in the air passages and in the airway led to the use of 1/100 gr. of atropine subcutaneously in repeated doses. Because of the cyanosis and rather feeble and shallow respirations, all sedation was omitted. By afternoon, the patient seemed better, although edema of the extremities had appeared. By 8 p.m., respirations were stronger, and he occasionally sighed heavily. Cold packs were again adminstered, and there was a drop of 2°F. in temperature.

Throughout the morning of the next day, the patient began to come out from under the previous sedation, and convulsions returned in rapid succession. Cyanosis, labored respirations, made noisy by secretions in the bronchi, and a pulse that was too rapid to count made us believe that he was in a very critical state. Sodium Amytal, 3¾ gr. every 3 hours, was resumed, and a transfusion of 500 cc. of citrated blood was given slowly. Edema persisted and interfered with venipunctures. By evening, the patient was quieting down and seemed further improved after a lumbar puncture and drainage at 10 p.m., but an attempted intravenous injection again precipitated a severe convulsion. The total medication in these 24 hours was 22½ gr. Sodium Amytal and 1/75 gr. atropine.

On July 11, the convulsions lessened in duration and intensity during the day, and for the first time the nurses did not administer ether during the seizures. At noon, the patient slept for 2 hours, and after this he passed gas and voided. Later, there were three convulsions that required the administration of ether. Edema continued, so that 1000 cc. of 5 per cent glucose in distilled water was given intravenously. Up to 9 p.m., 22½ gr. Sodium Amytal and 1/100 gr. atropine were given.

No sedative was given on July 12 until the patient suddenly had a severe convulsion at 8 a.m. This required ether. Several muscle spasms continued, and at 2 p.m. a lumbar puncture was done again and the pressure lowered from 200 to 75 mm., with immediate and striking improvement. No food or fluids had been taken by mouth up to this time, and at 10 p.m. the patient was therefore etherized and a nasal catheter passed through the left nostril into the stomach; hourly feedings of 45 cc. of an equal mixture of milk and lime water were begun. Acidosis was suspected, but we were heartened to find that the blood chloride was 99 milliequiv. per liter (normal, 100 to 105 milliequiv.), and the carbon dioxide combining power 24.3 vol. per cent. The nonprotein nitrogen was 27 mg. per 100 cc., the red-cell count 4,630,000, and the hemoglobin 15.6 gm. Throughout this day, the edema subsided, and the patient received 18¾ gr. Sodium Amytal and 6 gr. sodium luminal after 8 a.m.

The next day was encouraging, since there were only a few severe convulsions, and by evening there were only short spasms of the back, chest and leg muscles. The large amount of mucus in the throat and airway forced us to continue the use of frequent suction of the upper air passages and the repeated use of atropine. The total intake of milk and lime water by gavage was 1480 cc. Sedation totaled 11 gr. of sodium luminal and 11¼ gr. of Sodium Amytal.

On July 14, the patient seemed quieter until 5 a.m., when he vomited, became cyanotic and had a severe convulsion. The nurse then noted a diffuse, dull-reddish, morbilliform eruption. The fever and respiratory rate increased, and the pulse could not be felt. The jaws were still tightly locked, and the body was becoming cyanotic. The situation was precarious, and we feared that the patient would not survive the shock of serum sickness. At 11 a.m., we finally succeeded in completing a lumbar puncture, but we were surprised to find a normal initial pressure — 150 mm. The only treatment through this period was continuous oxygen by nasal catheter and repeated cold packs to the entire body. An intravenous injection of 500 cc. of 5 per cent glucose in distilled water was given in the hope of preventing edema. Feedings of milk and lime water through the nasal tube were continued. Sedation in this 24-hour period was 11 gr. sodium luminal and 7½ gr. Sodium Amytal.

On July 15, the worst seemed over, and by 3 a.m. the pulse was palpable. Cyanosis continued throughout the day, and the pulse became very thready in the afternoon; we therefore decided to transfuse him, 300 cc. of whole blood plus 150 cc. of saline being given. By 10 p.m., the patient looked better and breathed more easily, and the pulse was of better quality. A bloody nasal discharge began, and the nasal oxygen catheter was removed. He seemed weak and tired, and this was the first day without convulsions — there were only spasms of the muscle groups. The total sedation in these 24 hours was 12 gr. sodium luminal and 3¾ Sodium Amytal.

On the following day, the nurses were encouraged, and for the first time admitted that the patient might recover. He was noisy, cried out, and had an excessive amount of bloody mucus in the upper air passages. There were no spasms from 3 until 10 p.m. A high-calorie feeding mixture was started, and he was given 30 gr. chloral hydrate twice through the feeding tube. In this period, only 3 gr. of sodium luminal was given.

On July 17, the patient continued to groan and have muscle spasms, and the teeth could be separated about 1.0 cm. Frequent aspiration of the upper air passages was still necessary because of excessive mucus secretions. He was controlled with two 15-gr. doses of chloral hydrate through the feeding tube and 30 gr. once by rectum.

On July 18, fetor and a bloody nasal discharge about the feeding tube, which had been in place for 6 days, caused some concern. It was removed, and careful aspiration, followed by argyrol in the nose, preceded its reinsertion through the right nostril. A furuncle appeared on the left shoulder, so that the patient was turned every hour. Throughout the day, he was restless, the eyes moved, and he cried out; at 9 p.m., he first answered "Yes" and squeezed the nurse's hand. The nasal tube was removed at 5 p.m., and from then until August 2 it was put down each morning for the gavage feedings throughout the day and removed about 9 p.m.

The patient continued to have spasms about every two or three hours on July 19, but he was slowly gaining consciousness and voided in the urinal. He perspired freely, and mild dehydration was balanced with an intra-

venous injection of 800 cc. of 5 per cent glucose in saline. The sedation in these 24 hours totaled 5 gr. of sodium luminal and 30 gr. of chloral hydrate by rectum.

On July 20, the condition was about the same, and the patient was trying to talk. Much mucus secretion was aspirated from the upper air passages. An abscess on the scalp was noted. The sedation was 12 gr. sodium luminal.

From July 21 to August 1, the chief problem was a sudden, rapidly spreading furunculosis and cellulitis of the head and neck. The scalp was shaved, individual abscesses were drained, and alcohol and iodine were applied thrice daily. This treatment was painful but effective, although a large cervical abscess had to be drained under ether anesthesia on July 28. Likewise, pressure areas on the shoulders and back demanded constant nursing attention. The amount of feeding by tube was gradually increased, and the patient tolerated high-calorie mixtures, orange juice and 20 mg. of vitamin B daily. On July 24 and again on July 30, a loose tooth was discovered and removed. On July 24, some concern was caused by signs of congestion in the right upper lung, and an x-ray film of the chest disclosed a small dense area in this region. This, however, rapidly cleared, and on July 30 the chest film was normal. There was no evidence of a fracture of the ribs or vertebras. By July 27, the patient was able to swallow sips of water, but tube feeding had to be continued. From July 25 to 31, he was often disorientated, screaming and having hallucinations. Spasms of the muscles continued to keep the back arched, and we were unable to extend the legs completely. The sedation used in this period was chiefly sodium luminal subcutaneously, usually 3 to 6 gr. at night.

August 1 to August 29 marked the true period of convalescence in which each day showed a little gain in the general condition. August 2 was the first day in which the patient swallowed small amounts of soft food and could speak clearly but slowly. By August 5, he enjoyed having a book read to him for a short time. On August 6, another cervical abscess was drained, and on that day he listened to the radio and also fed himself.

On August 7, the red-cell count was 4,330,000, and the white-cell count 10,100. By August 14, the red-cell count had dropped to 4,000,000, and 4 gr. ferrous sulfate was given twice a day. On August 19, the first hour in a wheel chair caused a marked response, with fever, elevation of the pulse and fatigue. A swollen right axillary node began to cause pain and increased in size despite local applications of heat; on August 22, it was incised and drained. On August 27, the patient received 1.0 cc. of tetanus toxoid subcutaneously. After this, improvement was rapid, and he was discharged home on August 29.

On December 3, he was seen in the office and was given a second injection of 1.0 cc. of tetanus toxoid. At this time, he appeared entirely healthy and was mentally alert. He returned to school on January 6, 1941.

Discussion

Several features of this case warrant emphasis. The first is that the original injury occurred on the beach, an unlikely place for tetanus bacilli; one of us (R. H. M.)[1] had previously written that "ordinary wounds acquired in the home or in clean places, free from possible fecal contamination, or while bathing at the shore should not require tetanus antitoxin." Obviously, we can no longer regard the seashore as safe from tetanus infection. As Moore and Singleton[2] have recently pointed out, this disease is present all over the United States, but its geographical distribution is highest in the South and least in New England. Despite this reassurance to those in the cooler climates, it is known that tetanus causes over a thousand deaths yearly in this country.

The second feature is that the onset of tetanus six days after the injury indicated a poor outlook. The direct relation between the incubation period and the mortality in this disease has been known since the time of Hippocrates, but the recent statistics of Kirtley[3] are of interest. He found that 72 per cent of those developing tetanus within ten days of the injury died in one to sixteen days; death followed in only 18 per cent of those developing it ten days or more after the injury. The short incubation period in our case is indicated in Figure 1.

In the treatment of this case, we employed six principles that we believe were of almost equal value in restoring this boy to health: profound sedation, surgical removal of the focus of tetanus, moderate intravenous dosages of tetanus antitoxin, frequent lumbar punctures, maintenance of adequate respiratory exchange and the most intelligent nursing care.

Adequate sedation is of utmost importance, and we agree with Spaeth[4] that enough sedation to relax the patient should be given before operating to remove the focus of tetanus bacilli. In this way, one avoids precipitating severe and possibly fatal convulsions early in the disease. The sedatives that we chose were sodium luminal, Amytal and chloral hydrate in large doses, but Evipal, Pentobarbital and others are probably equally effective. We believe that morphine and other respiratory depressants are contraindicated because death may occur from respiratory embarrassment. Likewise, we cannot recommend avertin, magnesium sulfate, paraldehyde or other drugs known to cause occasional constitutional reactions. Curare and similar substances are not yet generally available, although the use of curare has been reported by Cole[5]; evidently, its therapeutic and toxic levels are dangerously close to each other.

The second principle is the need for the complete surgical removal of the focus of infection, which can best be done under general anesthesia. The wound should be left open and allowed to heal from the bottom. The topical use of sulfapyridine or another sulfonamide is probably of value in sterilizing the wound.

A most encouraging and instructive experience comes from Africa, where Bryant and Fairman[6] observed that the natives demanding the same treatment for tetanus as for cerebrospinal meningitis were given lumbar punctures and enough Evipal to keep them anesthetized for three or four days. In these cases, tetanus antitoxin was *never* given, whereas in some of the later cases It occurs to us that such a procedure should also delay the onset of serum sickness until the patient is better able to withstand it. In treating children, Dietrich[9] urges a slow but safe way to give serum antitoxin — the intramuscular route. Although our patient received an intrathecal injection of 10,000 units of antitoxin, it was done against the advice of one of us (R. H. M.). Since

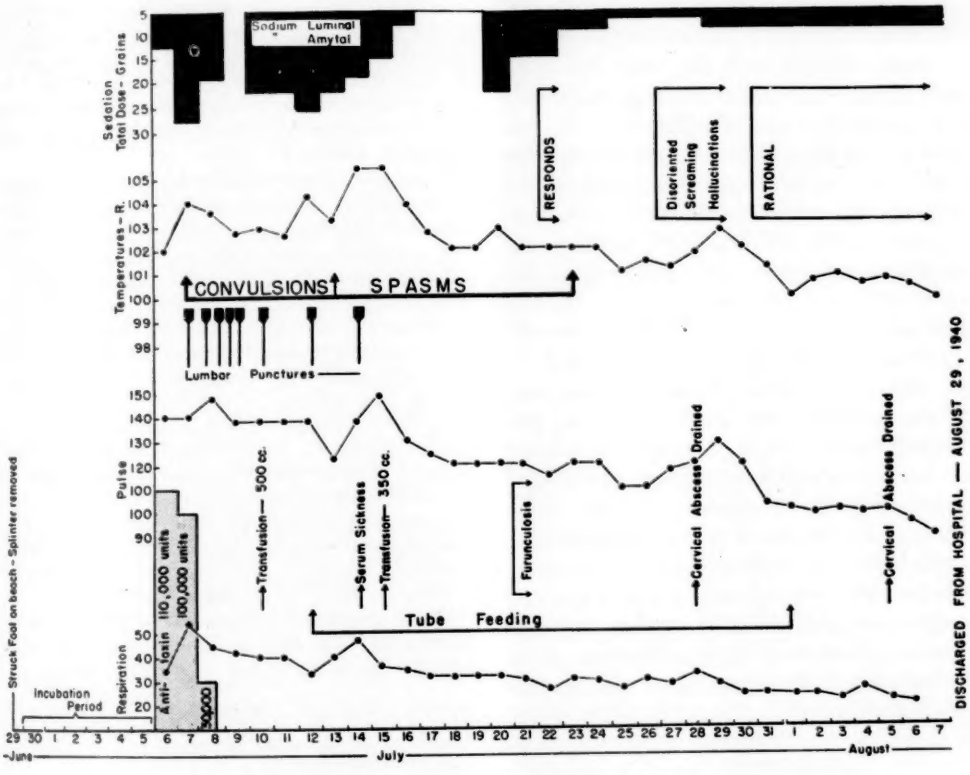

FIGURE 1. *Diagrammatic Summary of Data.*

sulfapyridine was administered. Unexpected recoveries were made under this therapy, and in the last 22 cases so treated only 5 patients died, which is a lower mortality than most reported series from this country.

This experience of Bryant and Fairman throws into sharp relief the problem of whether to give antitoxin, and if so, by what route it should be given and the optimum amount to give. We advocate a moderate dose about the wound and intravenously with physiologic saline for the first few days of the disease. It seems unnecessary to give enormous doses, for it has been shown experimentally that if one full lethal dose has reached the spinal cord, a million neutralizing units of antitoxin do not save the animal.[7] The daily injection of 5000 units of antitoxin has been recommended by Firor[8] to ensure the maintenance of an adequate titer of antibody in the blood stream. then, the reasons summarized by Hall[10] make this route seem quite inadvisable.

The next principle, possibly the most important one in our treatment, was the frequent drainage of spinal fluid. This procedure seems not to have been emphasized in previous reports. It is known that the cerebrospinal fluid is under an increased pressure during profound sedation and also during convulsive seizures; we therefore reasoned that lumbar puncture might bring relief to a patient in severe tetanus. The prompt alleviation afforded by these procedures seemed mechanical, allaying the nervous tension and convulsions.

The maintenance of a clear airway, an adequate supply of oxygen, the control of bronchial secretions and other measures such as the care of the skin, frequent turning, careful feeding by tube and the many details of the sickroom were largely the responsibility of the nurses.

Finally, we wish to direct particular attention to the use of tetanus toxoid. This should be used not only as a part of routine immunization in childhood but also at the time of injuries suspected of introducing tetanus bacilli. The occurrence of tetanus after the usual prophylactic dose of antitoxin is well known, and the recurrence of tetanus in the same patient has likewise shown that the disease itself may not confer immunity. Individual immunity may be linked to the occasional harboring of tetanus bacilli in the gastrointestinal tract, and it was the work of Ten Broeck and Bauer[11] that led to the suggestion that immunization could be established by the oral route. The use of toxoid, however, is far simpler and, according to Hall,[12] is completely safe if the toxoid is properly prepared. However, anaphylactic reactions, without fatalities, have been reported from England[13] as occurring twice in 64,000 men immunized for the Royal Air Force. It was found that these 2 persons were sensitive to the Witte's peptone used in preparing the toxoid.

Ramon, the discoverer of toxoid, has recently advised[14] the treatment of acute tetanus as follows: 2 cc. of toxoid subcutaneously and 150,000 units of antitoxin intravenously immediately, and then increasing doses of toxoid every five days, so that the patient receives 4 cc. on the fifth day, 6 cc. on the tenth day and so forth. Such treatment may be effective, but we remain convinced that the constant attention of the nurse and doctor and the employment of the six principles given here are essential to the recovery of the patient severely ill with tetanus.

Summary

A successfully treated case of tetanus, developing six days after injury, is described in detail.

Six principles of treatment that seem essential to recovery are discussed.

Recently, we have successfully treated another patient using these same principles. The summary of the case, reported through the courtesy of Dr. P. J. Shannon, of Hingham, Massachusetts, is as follows:

On May 27, 1941, a 14-year-old girl cut the dorsum of her right foot on a sickle that was rusted. She was seen within 10 minutes, and the wound cleansed with hydrogen peroxide and Merthiolate. Eight dermal sutures were used to close the wound. Three days later the wound discharged pus, and hot boric acid soaks were applied. On the 4th day fever developed, so sulfathiazole, 1 gm. every 4 hours, was given for the next 4 days. Hydrogen peroxide dressings to the wound were continued; she seemed improved and was active.

On June 6, 9 days after the injury, she first developed trismus, and by evening she had back pains, headache and malaise; she was given 1500 units of tetanus antitoxin intravenously. The symptoms and signs of tetanus progressed through June 7, and on June 8 she was gravely ill and admitted to the Cohasset Hospital. In the next 24 hours she received 140,000 units of antitoxin intravenously and was given repeated doses of ⅙ gr. of morphine. At two periods in the late afternoon she became cyanotic and choked with mucus during the seizures. By evening of June 9, when first seen by one of us (E. M. C.), she was critically ill with severe tetanus and was having generalized spasms or seizures about every 3 to 5 minutes. Local tetanus also caused contractures of the right foot and leg. A culture of the wound was taken immediately and eventually yielded *Cl. tetani*.

Twenty thousand units of antitoxin was injected into three separate areas about the wound before it was débrided and left open. Sulfapyridine was dusted into the wound. Lumbar puncture was done, and dramatic relief of symptoms came after drainage of a clear fluid estimated to be under an initial pressure of 500 mm. Sedation was changed to sodium luminal, about 2 gr. subcutaneously every 3 hours. The nursing care was under the supervision of one of the nurses who had cared for the first patient.

In the subsequent 9 days in the hospital, this girl improved gradually. The lumbar puncture was repeated on June 11, with obvious relief of symptoms. The temperature fell slowly to normal from a level of 102 to 103°F., and the pulse declined gradually from a level of 120. By June 14, swallowing was less difficult and she had regained control of the sphincters. Tetanus toxoid was given as follows: June 12, 1 cc.; June 13, 2 cc.; June 14, 3 cc. Sedation was gradually reduced from a daily total of 16 to 6 gr. On July 18, for economic reasons, she was returned to her home for convalescent care.

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MEDICAL PROGRESS

GENERAL ANESTHESIA*

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ALTHOUGH inhalation anesthesia is usually considered synonymous with general anesthesia, other routes of administration of anesthetic drugs must be considered in a discussion of this subject.

RECTAL ANESTHESIA

Certain drugs are administered by the rectal route to produce general anesthesia. The most commonly used of these are tribromethanol (Avertin), paraldehyde and the barbiturate, Evipal. Of these, Avertin is probably the best known. These drugs are neither satisfactory nor safe when used alone, but are valuable as adjuncts to other anesthetic agents. They are useful as supplements to nitrous oxide and oxygen when a noninflammable inhalation anesthetic is desired. The administration of one of these drugs in the patient's room is often of great value in allaying the fears of nervous patients. Their principal disadvantages are that proper dosages cannot always be determined, owing to varying rates of absorption from the intestine, and that their administration is often followed by a prolonged period of depression. Because of the prolonged action of these drugs, respiratory obstruction is prone to occur during the postoperative recovery period.

INTRAVENOUS ANESTHESIA

The intravenous method of administration of anesthetic drugs has increased in popularity with the introduction of the shorter acting barbiturates. The most commonly used of these are Evipal Soluble and Pentothal Sodium. Their principal advantages are ease of administration and an accuracy of dosage comparable with that of the inhalation anesthetics. Furthermore, induction is usually rapid and pleasant, and recovery as a rule is free from such postoperative discomforts as nausea and vomiting. In common with the rectal anesthetic drugs, they are free from fire hazard.

On the other hand, the intravenous barbiturates have certain disadvantages of which the anesthetist should be cognizant before employing them as anesthetic agents. The margin of safety (the range between the anesthetic dose and the lethal dose) is relatively narrow. Reaction to comparable doses varies widely, even among patients of the same age and of apparently equal vigor. Since these drugs are largely broken down in the liver and eliminated through the kidneys, they should not be used in patients with impaired liver function or uremia. Because of the means by which the barbiturates are eliminated, there tends to be an accumulation of the drug in the body during prolonged anesthesia. After the anesthesia has been carried on for some time, the addition of very small amounts of these drugs may push the level of anesthesia to a dangerous depth. Under intravenous barbiturate anesthesia, hiccups occur fairly frequently. This is a troublesome complication, the treatment of which is often quite unsatisfactory. The natural tendency when they occur is to increase the depth of anesthesia; this is usually ineffectual and may be hazardous. When anesthesia is light, laryngospasm is prone to occur if the larynx is irritated by operative manipulation or by secretions. For this reason, these drugs are usually not the anesthetics of choice for laryngoscopy and bronchoscopy. In patients with impaired kidney function, recovery may be somewhat prolonged. Finally, many patients, especially those in the elderly group, may be mildly disorientated for a period as long as twenty-four hours after awaking.

The intravenous injection of barbiturates must be done with care. If a 5 per cent solution of Pentothal Sodium is inadvertently injected into the subcutaneous tissues, marked irritation is likely to occur, and a solution as concentrated as 10 per cent may produce a severe slough. Furthermore, as pointed out by Lundy and Adams,[1] delayed phlebitis may follow the intravenous injection of a 5 per cent solution of Pentothal. For this reason, it is advisable to use a 2.5 per cent solution of this drug intravenously.

A simple and inexpensive apparatus has been described by Nicholson and Sise[2] for the administration of intravenous anesthetic agents. It consists of a small-caliber, thick-walled rubber tube about 75 cm. in length, with a glass observation tube on one end — to which the needle is fitted — and a stopcock with an adapter for a syringe on the other.

Intravenous barbiturate anesthesia is valuable for short operations when little muscular relaxation is required. It is particularly useful when

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high-frequency electrosurgical apparatus is to be used or when the operative procedure is to be carried out in the x-ray room.

These drugs have recently been used as complements to spinal anesthesia. They should be used only for the comfort of the patient and never to augment a spinal anesthetic that is inadequate or has terminated; an inhalation anesthetic is preferable for this purpose.

Inhalation Anesthesia

Despite the increase in the use of other routes for the administration of anesthetic drugs, the inhalation method is probably still the most widely employed and the safest in general use, and is certainly the most flexible.

Most drugs used by this method are eliminated largely through the lungs, the liver and kidneys playing a minor part in their removal from the body. Increasing concentrations of the drugs in the blood bring about fairly well-marked changes in the character and rate of respiration, making guideposts by which the anesthetist may judge the depth of anesthesia. The flexibility of this method is such that an anesthetic course may range in depth from mere insensibility to complete flaccidity of most of the skeletal muscles, and in length from a fraction of a minute to many hours. Apparatus for the inhalation method may range from a handful of gauze and a container of anesthetic drug to the most complicated precision gas machines.

Today, although ether is still one of the most satisfactory and widely used general anesthetic drugs, there are many other well-established agents such as cyclopropane, ethylene, nitrous oxide and divinyl ether, which may be administered by the inhalation route, either alone or in combination with ether.

Divinyl ether has a rather limited use. It is a short-acting agent, and when given by the open-drop method is useful when abscesses are to be opened or packs removed or when other brief procedures are to be carried out. Prolonged divinyl ether anesthesia with inadequate oxygen may produce liver damage.

A new anesthetic agent, cyclopropyl-methyl-ether, has been introduced,[3] but its use is still in the experimental stage. Its sponsors state that it is more potent than ether and may possess many of the advantages of cyclopropane.

Cyclopropane, because of its many advantages, has attained great popularity in the few years it has been in use. In many ways, it can still be considered the closest approach to an ideal anesthetic agent that has yet been found. Its extreme potency allows for rapid induction and a very high concentration of oxygen in the anesthetic mixture. The high concentration of oxygen reduces respiratory activity during anesthesia, but oxygenation has been shown to be adequate.[4] Its effects on the chemical constituents of the body are slight, and it is rapidly eliminated when the anesthesia is terminated.

Cyclopropane, however, has certain properties that prevent it from being an ideal anesthetic agent. Like many other hydrocarbons, it is inflammable. Unmixed with other anesthetic agents, it rarely produces the degree of relaxation required for abdominal surgery unless concentrations are used that are generally accepted as being outside the bounds of safety. Methods have been described and are in use in which concentrations as high as 75 per cent cyclopropane in oxygen[5] are delivered to the patient, and the subsequent apnea handled by artificial respiration or passive breathing. With this technic, the concentration of anesthetic drug is such that the respiratory center is itself anesthetized, and adequate oxygenation is maintained by the pumping of the anesthetic mixture into the lungs by the anesthetist. Relaxation is reported to be as complete as that obtained with spinal anesthesia, and although cardiac irregularities may be observed during induction, they are not found when the required depth of anesthesia is reached. This method is not in general use and seems to be more than a little hazardous except in the hands of those who are highly experienced with it.

Early in the use of cyclopropane, it was found that under fairly deep anesthesia cardiac irregularities developed in a certain number of patients. These are likely to occur with thyrotoxic patients and with those who have impairment of the mechanism of cardiac conduction. It was also found experimentally and, unfortunately, in the operating room that the injection of adrenalin into an animal or patient under cyclopropane produces cardiac irregularities and, in some cases, cardiac arrest probably because of ventricular fibrillation.[6] Laboratory studies on dogs have shown that the irritability of the automatic tissue of the heart is increased by cyclopropane and that morphine tends to increase this irritability further; the barbiturates decrease this tendency to cardiac irregularities to a point where, with deepening anesthesia, respiratory depression precedes their onset.[7] Similar laboratory studies have shown that procaine in small amounts, administered by the intravenous or intracardiac route, arrests the ventricular fibrillation that has been induced by superimposing adrenalin on cyclopropane, and restores normal rhythm.[8] Since procaine is extremely toxic intravenously, it is impractical for clinical use to

prevent cardiac irregularities. Although morphine seems to increase the tendency to cardiac irregularities, and occasionally to subsequent ventricular fibrillation in the dog, it is an extremely useful preoperative drug. Clinically, the barbiturates are not so satisfactory for preoperative medication when used alone as when combined with morphine. Preoperative medication appears to lengthen the margin of safety of cyclopropane by reducing the amount required to produce anesthesia without a comparable reduction in the amount necessary for respiratory arrest.

The majority of cardiac irregularities due to cyclopropane can be eliminated by lessening its concentration in the anesthetic mixture. However, it may be necessary to add another drug to maintain adequate depth. If care is taken that the cyclopropane concentration does not exceed 10 per cent, perceptible cardiac irregularities can usually be eliminated. If this concentration is inadequate to maintain anesthesia, ethylene or ether may be added as a supplement. When ethylene is used as the supplementary agent, the concentration of oxygen must be kept above 20 per cent, or else one of the advantages of cyclopropane, namely, a high concentration of oxygen, is eliminated. A combination of 60 per cent ethylene, 30 per cent oxygen and 10 per cent cyclopropane will give a mixture that is, in most cases, adequate for anesthesia and allows sufficient oxygen. Recovery, when cyclopropane and ethylene mixtures are employed, seems to be as rapid as when either of these drugs is used alone.

Cyclopropane may be used instead of ether to supplement ethylene and oxygen or nitrous oxide and oxygen anesthesia. This allows for the administration of adequate oxygen at all times, in addition to the rapid induction and recovery characteristic of gas anesthesia. Although a mixture of ethylene and oxygen permits a somewhat higher concentration of oxygen (15 per cent or less) than that of nitrous oxide and oxygen (10 per cent or less), this is still too narrow a margin to ensure adequate oxygenation. This margin may be further narrowed if the patient has an increased metabolic rate. It must be borne in mind that the absence of cyanosis cannot always be taken to indicate adequate oxygenation.

Although a mixture of nitrous oxide and oxygen is usually inadequate to produce satisfactory anesthesia without suboxygenation, it has the advantage of being noninflammable. Combined with some nonvolatile anesthetic agent, such as intravenous Pentothal or rectal Avertin, it may be used with safety when x-ray or high-frequency electrical apparatus is employed. This combination also permits the use of the cautery about the head and neck.

The explosion and fire hazards present with most inhalation anesthetics are of sufficient importance to require special consideration. Precautions against these hazards should be most careful and painstaking. Ether, cyclopropane and ethylene, when mixed with oxygen, are potential bombs and should be handled with the same degree of respect.

Obviously, no open flames should be allowed in the operating room. If spark-proof light switches and wall plugs are not available, all electrical connections should be made and turned on before the anesthesia tanks are opened, and should remain on until the anesthesia is completed and the machine flushed out with air. This flushing of the apparatus can be done by the anesthetist, blowing through the breathing tubes. Electrical equipment in the operating and anesthesia rooms should be carefully and frequently inspected by a qualified electrician. Electric motors and pumps should be placed outside the room when tanks of anesthetic gas are open. If high-frequency electrical apparatus is to be used, ethylene, cyclopropane or oxygen and ether should not be administered.

Ether vapor mixed with air probably does not present so great an explosion hazard as ether vapor mixed with oxygen, ethylene and oxygen or cyclopropane and oxygen. Experiments done by Dr. J. W. King,[9] of the Massachusetts Institute of Technology, have shown that no mixture of ether and air will explode with the violence that some mixtures of ether and oxygen do or with the violence that all explosive mixtures of cyclopropane and oxygen do. However, when ether in air is being used in the presence of high-frequency electrosurgical apparatus, special care should be taken to deliver the exhaled gases at a point several feet from the site of operation and from the electrical apparatus.

Despite the above precautions, the greatest source of ignition in the operating room, namely static electricity, still has to be dealt with. Obviously, the ideal method for dealing with this hazard would be for every piece of apparatus and all persons in the operating room to be electrically connected. This is not feasible at the present time. If the patient, anesthetist and apparatus (gas machine and operating table) nearest to the explosive anesthetic mixture are electrically connected, a great factor of safety is introduced.[10, 11] The Horton intercoupler* is an apparatus de-

*Manufactured by Technequipment Company, Brookline, Massachusetts.

signed to connect electrically these four objects in the danger zone.

Inflammable gases should be given in a closed system with carbon dioxide absorption whenever possible, and the room should be adequately ventilated so that leakage of potentially explosive gases may be quickly dispersed. Some work has been done on the quenching value of inert gases such as helium[12] and nitrogen[13] on explosive gaseous mixtures. This may offer an additional safeguard; however, it is still in the experimental stage.

During the administration of a general anesthetic, attention to the maintenance of an adequate airway is as essential as the selection of the anesthetic agent. Nasopharyngeal and oropharyngeal tubes of various kinds relieve respiratory obstruction from the lips and tongue. Complete control of the patient's airway can be ensured only through the use of an endotracheal catheter. By this means, obstruction at the larynx is eliminated, and pressure on the trachea, especially in surgery about the neck, does not result in obstruction to respiration. By means of an endotracheal tube, obstruction from mucus, vomitus and blood may be avoided, since they may be removed by suction. The topical application of 10 per cent cocaine by means of an atomizer allows introduction of an endotracheal tube with the patient in a fairly light stage of anesthesia and eliminates considerable spasm and coughing when the tube comes in contact with the tracheal mucosa. In patients with marked tracheal obstruction, intubation may be done with topical anesthesia alone, and general anesthesia can be induced after the tube is in place.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27431

PRESENTATION OF CASE

A sixty-two-year-old sheet-metal worker was admitted to the hospital because of intermittent pain in the abdomen of one week's duration.

For the preceding six to eight years, the patient had been troubled with flatulent indigestion, mild constipation and aversion to fatty foods. On several occasions, he had experienced colic. Nine months before entry, he developed severe knifelike abdominal pain, accompanied by jaundice, and had been seen in another hospital, where cholecystectomy and exploration of the common bile duct were performed. The gall bladder was enlarged and thickened, and the common duct was found dilated, with a number of medium-sized faceted stones in its distal end. The liver was described as "mottled." The convalescence was not remarkable, drainage being discontinued on the eighteenth day. The operation seemed to relieve the symptoms except for the intolerance to fatty foods.

Ten days before entry, closely following a meal of stewed chicken, a moderately severe aching pain spread across most of his abdomen. This pain was different from any that the patient had felt in previous attacks in that it seemed more constant and severer, and occurred lower in the abdomen. It continued with only slight remissions until entry. Three or four days before admission, there was gradual onset of jaundice. Increasing amounts of gas were passed by mouth and by rectum. The night before he came to the hospital, the patient vomited slate-colored fluid a dozen times, with some relief.

The past history was not significant. His first wife and his son by her had both died of tuberculosis.

On examination, the patient appeared acutely ill and definitely jaundiced. A right upper abdominal scar was present. The abdomen was tender, especially in the right upper quadrant. The liver was slightly enlarged and tender. External hemorrhoids were present. The only other positive findings were slight enlargement of the heart and a few basal rales. The blood pressure was 198 systolic, 100 diastolic.

The temperature was 102°F., the pulse 70, and the respirations 30.

Examination of the blood showed a red-cell count of 4,000,000 with 60 per cent hemoglobin, and a white-cell count of 18,400. The van den Bergh reaction was biphasic, 11.1 mg. per 100 cc. The blood sugar was 128 mg. per 100 cc. (148 mg. when repeated). The prothrombin time was 26 seconds. The urine showed a +++ test for albumin, a +++ test for bile pigments and a trace of sugar, on repeated examinations. The stools were not reported.

A flat-plate roentgenogram of the gall-bladder region was negative. Duodenal drainage was performed, but no bile was found in the sample.

Operative intervention was deferred because of the patient's poor condition. The glycosuria and hyperglycemia were studied, but no specific therapeutic measures were taken.

During the first few hospital days, the patient felt improved. The fourth day, however, his temperature rose to 106°F. The liver edge became much more tender, and jaundice deepened. There were no chills or flank tenderness. A roentgenogram of the chest taken in the horizontal position showed the diaphragm to be high, with poor aeration of the lungs. A three-day course of sulfadiazine was given, following which the temperature dropped abruptly to 98°F. The drug was discontinued because of a fall in daily urine output to only 150 cc., accompanied by a rise in blood nonprotein nitrogen to 99 mg. per 100 cc. The patient became progressively more jaundiced. He was given high-carbohydrate therapy and intravenous amino acids. On the fifth hospital day, he vomited several times. Irregularity of the heart was noted. An electrocardiogram demonstrated auricular fibrillation, with a rate varying from 100 to 140 per minute; there were also irregular ectopic ventricular beats. Digitalis was therefore given, resulting in a return to fairly normal rhythm by the ninth hospital day. Four days later, the patient suffered a chill, and his temperature spiked to 104°F. During his last few days, the blood nonprotein nitrogen rose to 270 mg. per 100 cc. This was thought to be due to the amino acid therapy he was receiving, since his urinary output had been restored. He became comatose and died on the fourteenth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. RICHARD H. SWEET: I remember this patient. He came in on the ward a day or so be-

fore I went off service. I do not know what the autopsy showed. He was under the care of Dr. John P. Stewart, who had charge of the cases of common-duct obstruction at that time. My only recollection is that he was exceedingly ill.

We are dealing with an obese, elderly man who was obviously in poor condition at the time of entry. The evidence for that seems to me to be, first of all, the albuminuria, which is also, perhaps, evidence of kidney damage. He may have had diabetes. He had cardiac enlargement with auricular fibrillation and ectopic beats. He had rales in the chest and definite anemia. The second fact that comes to the fore is that he had a known history of gallstones and stones in the common duct removed at operation. The third point I should like to bring out is that he subsequently had a characteristic history of common-duct obstruction, due most likely, I should think, to a stone left in the common duct at the previous operation. I say that because I happen to be a surgeon. The history is very typical. The patient was relieved for a time and felt all right, then had sudden onset of pain, and within a few hours, jaundice and obvious evidence of obstruction. If he had been my patient, I should have thought I had left a stone in the common duct. Secondary to this common-duct obstruction, the record shows evidence of infection of the biliary tract, with chills and fever, the so-called "Charcot syndrome." I should expect that he had cholangitis of an acute nature, with secondary liver damage.

Certain aspects of the case represent complicating factors, chiefly the therapy. One notes that sulfadiazine was given. Then, shortly after that, the sulfadiazine was omitted because of a marked reduction in urinary output and a rise in nonprotein nitrogen. This may have been due to the chemotherapy. He may have had kidneys already damaged by arteriosclerosis. It is also known that people with this sort of liver disease do sometimes present evidence of renal damage presumed to be secondary to the liver insufficiency. The argument whether such patients die of liver disease or kidney disease is still a live one, in certain clinics at any rate. The other complicating factor is the amino acid therapy. I am not sure why amino acids were given. The record does not say whether the plasma-protein level was low, or whether he had generalized edema, suggesting perhaps nutritional lowering of the plasma protein; at any rate, he did have amino acid therapy, which I believe had been used very little up to that time on the service, and it probably did account for the marked rise of nonprotein nitrogen, if the determination was made at the time that the amino acids were circulating in the blood. At that time, the urinary output was not low, and I should assume that the amino acids were not a factor in his death, although that possibility should be kept in mind. I see no evidence for any other conclusion than that he had choledocholithiasis, a stone presumably left in the common duct, in addition to infection, acute cholangitis and, perhaps, liver abscess. He must have had renal damage, whether on the basis of arteriosclerosis or not I do not know. I cannot go so far as to call this a case of hepatorenal syndrome.

Clinical Diagnoses

Cholangitis, acute.
Multiple liver abscesses.

Dr. Sweet's Diagnoses

Choledocholithiasis, with obstruction.
Acute cholangitis.
Multiple hepatic abscesses?
Renal damage.

Anatomical Diagnoses

Cholangitis, with abscess formation and thrombosis of hepatic veins.
Subhepatic abscess, probably at site of amputated gall bladder and cystic duct.
Icterus, generalized.
Pulmonary collapse and congestion, bilateral.
Pancreatitis, minimal.
Bile nephrosis.
Decubitus ulcer, right buttock.
Obesity.

Pathological Discussion

Dr. Tracy B. Mallory: Some of the findings post mortem were quite obvious. The patient did have very extensive sepsis in the biliary tract, with a diffuse intrahepatic cholangitis and multiple liver abscesses. There was also a subhepatic abscess, in the immediate area of the removed gall bladder, a large, well-localized pocket of pus about the size of a distended gall bladder. There were no persisting stones in the duct; they had all been removed. The heart was somewhat hypertrophied, suggesting that he may well have had some hypertension and perhaps some renal insufficiency based on that, although the kidneys showed no striking degree of nephrosclerosis. At the time of autopsy, we were impressed with a little discoloration at the junction of the pyramids and cortex, which we thought might possibly be crystals of sulfapyridine, and in view of his terminal anuria we naturally attempted to establish that

point. We could not. There were no crystals, and I think the discoloration was due to bile-stained casts, rather concentrated at that point in the tubules. The kidneys were grossly swollen, and the cells of the convoluted tubules were likewise swollen and contained moderate amounts of bile pigment, the picture of what has been called bile nephrosis. That is something that one sees with considerable regularity in deeply jaundiced patients. So far as I can make out, its severity bears no correlation to the presence or absence of the so-called "hepatorenal syndrome." I should be tempted to think that this man died from the combination of sepsis and hepatic insufficiency, and rather discount any renal factor, although on anatomic grounds it is impossible to do so with certainty. I should think that with such extensive liver disease as this, one ought to use caution in amino acid therapy. We know that amino acids are deaminized solely in the liver, and in the face of a nonfunctioning liver I do not see how the organism can dispose of them.

DR. SWEET: It seems to me that that is the important thing about this case. As I read it over, I wondered what amino acids and the sulfadiazine had to do with his death. But he was an extremely sick man and probably would have died anyway of his underlying disease.

DR. MALLORY: So far as the anatomical evidence goes, we can rule out the sulfadiazine as the cause of renal insufficiency.

CASE 27432

PRESENTATION OF CASE

A sixty-two-year-old housewife entered the hospital because of repeated episodes of pain in the chest.

Nothing was known about the first attack, except that the pain had been mild and had involved the left chest approximately six months before entry. The second episode, two months before admission, consisted in a moderately severe pain in the left chest accompanied by an ache in the left elbow. There was no radiation of pain to the shoulder, and there was no respiratory difficulty. The patient's physician prescribed a course of theobromine, potassium iodide and phenobarbital. At the time of this attack, the blood pressure was 164 systolic, 104 diastolic, the pulse 84, and the temperature 98.1°F. The following day the blood pressure was 140 systolic, 80 diastolic, the pulse 72, and the temperature 98.3°F. On the third day, the blood pressure was 130 systolic, 90 diastolic, and the pulse and temperature remained as before. The patient was kept in bed for six weeks and then allowed up. The next day, she experienced minor chest pain. About ten days later, — ten days before entry, — pain again appeared, this time in the right chest, radiating to the shoulder and also across the abdomen. Three days before admission, there was an insidious onset of slight painless coughing. In the evening, a spell of severe coughing lasted six or seven minutes. During the night, the patient perspired freely and her heart seemed to "beat twice as fast as normal." The following day, she complained of a sensation "like a collection of gas" below the right breast, more disturbing when she coughed. Her local physician gave morphine and began a course of sulfathiazole.

The patient had had varicose veins for years and remarked that she had had a "sore spot" behind her left knee about ten days before she came to the hospital.

Physical examination showed an elderly and slightly obese woman, who was not acutely ill. She was pale, with cyanotic lips. Her respirations were grunting and cut short by pain, and there was definite splinting of the right chest. The breath sounds were diminished at the right base, with many crackling rales. The heart sounds were poor, although they seemed accentuated over the mitral area. The blood pressure was 120 systolic, 90 diastolic.

The pulse was 104, and the respirations were 28.

A roentgenogram of the chest showed the diaphragm to be high on both sides. In the left lung field, a band of density obscured the lateral shift of the diaphragm. On the right, there were numerous linear bands and some haziness just lateral to the hilus. The upper lung fields were clear, and the heart shadow was not displaced.

During physical examination, the patient suddenly turned ashen. Her lips became more cyanotic, and beads of perspiration stood out on her face. There was partial loss of consciousness. The respirations became more rapid. Morphine, atropine and Coramine were given, and the patient was placed in an oxygen tent. In the course of fifteen minutes, the respirations became slower and more shallow; death followed, an hour and twenty minutes after admission.

DIFFERENTIAL DIAGNOSIS

DR. RENO PORTER: Here is the case of a woman who, except for varicose veins, was in good health and feeling well until six months before admission. She subsequently experienced four attacks of chest pain. There were no other significant signs or symptoms, such as cough, respiratory dif-

ficulty and changes in pulse and temperature, and no change in blood pressure. The fact that the location of the pain varied from one attack to another tends to rule out many localized processes originating in the lungs themselves. The last attack was ten days prior to admission, when the patient also noted a sore spot behind her left knee. This is a very important point and gives some indication about the origin of most of her difficulties, namely, a phlebitis. However, one could suspect such an origin without this evidence, because pulmonary emboli usually occur repeatedly before the offending phlebitis becomes obvious. For three days before admission, the patient had symptoms indicative of pleurisy, with cough, splinting of the right side of the chest, pain on respiration and x-ray evidence of multiple lung lesions. Then she died abruptly, soon after she was admitted.

It is quite evident from the data presented that she had repeated pulmonary emboli. The terminal event was probably massive pulmonary embolism. I think one can conclude this from the repeated attacks of chest pain, a story quite characteristic of pulmonary infarction, and also from the fact that x-ray examination showed evidence of multiple lesions. One would, perhaps, like a little more evidence, such as symptoms and signs of respiratory difficulty, as well as temperature and pulse changes. They were not present, but their absence does not rule out the possibility, because embolism can occur without objective evidence.

The question in my mind is whether this entire story can be explained on the basis of pulmonary emboli. It has been generally assumed, and I for a time had the impression, that pulmonary emboli are nearly always secondary to something else, commonly following a surgical procedure or occurring as a complication of heart disease. Hampton and Castleman[1] have shown that this is not necessarily so by their study of 307 cases of pulmonary infarction. They found that in 30 per cent the patients were nonsurgical and noncardiac. This patient, we know, had not been operated on, so that there was no predisposing factor. The heart on physical examination was normal in size, with no significant murmurs. She did have hypertension, however. One cannot definitely rule out the possibility that one of these attacks of pain was coronary thrombosis. Her doctor apparently thought that she had had a coronary thrombosis, since she was kept in bed for six weeks. The onset of chest pain, however, is the only indication. There were no other striking signs or symptoms, nor is there an electrocardiogram to help us. There was apparently no significant drop in blood pressure. The blood pressure fell somewhat in bed, but this could be explained by bed rest alone. There is very little evidence on which to make the diagnosis of coronary thrombosis.

Other conditions that might have occurred, such as infection, which her doctor suspected because he gave her sulfathiazole, need not be considered here. She did at one time complain that her heart beat more rapidly than normal. One must think of the possibility of an arrhythmia producing emboli that might go to the lungs. This tachycardia occurred during only one attack of pain, with no subsequent reference to irregularity. I think that this can be dismissed as a simple reaction to any insult to the lung.

I shall make a single diagnosis of multiple emboli. They arose probably from a thrombophlebitis of the popliteal vein on the left and possibly from the varicosities she had on the right. The emboli were multiple, and there should be infarctions of the lung, both old and recent. She may have had some pleural effusion on the right, associated with infarction, and I think she died of massive pulmonary embolism.

Dr. Paul D. White: It is rare to find coronary episodes in the form of coronary thrombosis recurring repeatedly over a period of a few months. I therefore think that although pain was the characteristic symptom in the beginning in this case, the frequency of the attacks is very much against myocardial infarction as the explanation of the whole story. As Dr. Porter says, coronary thrombosis may have begun the sequence of events, but the later "heart attacks" were in all probability due to pulmonary emboli.

The hypertension we know very little about; there is simply a record of a blood pressure of 164 systolic, 104 diastolic, at the time of the attack. This may have been a rise, the result of pain or discomfort, but on the other hand, the pressure might have dropped to this level; so that myocardial infarction might have been present despite the relatively negative examination. We do not have a definite statement about the heart size, simply the signs; we should expect, however, that the x-ray report would have shown cardiac enlargement. Tachycardia is common in pulmonary embolism. It may be the only symptom of an attack of pulmonary embolism, and paroxysmal tachycardia may be wrongly diagnosed to explain such episodes as occurred in this case, which I think Dr. Porter has correctly interpreted.

Clinical Diagnoses

Recurrent coronary thrombosis.
Pulmonary embolism.

Dr. Porter's Diagnoses

Thrombophlebitis, left popliteal vein.
Multiple pulmonary emboli.
Pulmonary infarcts, old and recent.
Massive pulmonary embolism.

Anatomical Diagnoses

Coronary thrombosis.
Myocardial infarction.
Thrombosis of popliteal vein, left.
Multiple pulmonary infarcts.
Acute massive pulmonary embolism.

Pathological Discussion

Dr. Benjamin Castleman: This patient had, as Dr. Porter predicted, numerous infarcts in the lung. They were present in every lobe of the right lung, more numerous in the lower lobe, and were of varying sizes and ages. There was no pleural fluid, but the pleura was fibrinous over the lower lobe. The main branches of the pulmonary artery were filled with emboli, and one large embolus was present in the right ventricle, extending through the pulmonary valve. The source of the emboli was in the left leg — the popliteal vein and many of its branches and tributaries were completely filled with thrombi. The heart weighed 375 gm. and showed slight hypertrophy. The coronary arteries showed severe sclerosis, with occlusion and narrowing. The left descending branch was almost completely occluded by a grayish thrombus, which might have been two or three months old. The anterior wall of the left ventricle was infarcted, and according to the criteria that were reported a few years ago by Mallory, White and Salcedo-Salgar[2] on the various ages of myocardial infarcts, was about two months old. This would correspond with the second episode. I therefore suppose that the patient started off, as Dr. White suggested, with a coronary thrombosis, was kept in bed for six weeks, allowing the peripheral thrombi to form while she was in bed, and when she got up developed a series of emboli and infarcts. Is it a general procedure to exercise the legs of patients with coronary disease?

Dr. White: Rest in bed for two months is much too long for the average person with acute coronary thrombosis and myocardial infarction, particularly because of the danger to the general health, especially from thrombophlebitis. There are, to be sure, some patients with congestive failure or other complications who may have to stay in bed longer, but as a rule, for patients with average-sized infarcts three or four weeks in bed are enough. Some have thought me unwise in advising such a short stay, but experience has confirmed its adequacy, in fact its preference. The chair position, incidentally, is better than the bed position so far as the heart is concerned. Instituting activity as soon as possible aids the peripheral circulation, as well as the morale and general health. I suppose we all tend to neglect to maintain adequate peripheral circulation in bed patients, which we can do by massage and even by active leg exercise. After the first two weeks, as soon as danger from the acute heart attack is largely over, the leg muscles need massage or exercise.

Dr. Howard B. Sprague: I have done that routinely for years. As soon as the fever is over, I start leg massage and encourage the patient to move the feet and wiggle the toes.

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ENCEPHALITIS AND THE HORSE

Once again, the importance of animal reservoirs of diseases affecting human beings comes to the fore. Recent work[1-4] from the West indicates that the virus of the St. Louis type of encephalitis is present in certain wild and domestic birds and mammals.

Especially in the group of encephalitides due to specific viruses has the role of animals become increasingly evident. About six months ago, Webster[5] pointed out that no less than nine distinct viruses — those of rabies, poliomyelitis, lymphocytic choriomeningitis, Japanese B encephalitis, louping ill, Australian X disease, virus B, equine encephalomyelitis and St. Louis encephalitis — are known to produce primary encephalitis in man. At that time, there was good evidence of mammalian or avian host reservoirs for six of these agents. The virus of rabies was demonstrated in animals in 1881. During the last decade, the virus of lymphocytic choriomeningitis was found in the mouse, that of louping ill in sheep, that of virus B in the monkey, and that of equine encephalomyelitis in horses and birds. Furthermore, the serums of sheep and horses have been found to contain neutralizing antibodies for the virus of Japanese B encephalitis. All these diseases were thus linked with animals. Only three viruses — those of poliomyelitis, Australian X disease and St. Louis encephalitis — gave no evidence of potential animal reservoirs.

Now, however, the virus of St. Louis encephalitis, as well as that of the Western type of equine encephalomyelitis, has been incriminated in both horses and human beings in the Yakima Valley of Washington by the work of Hammon,[1] and in Weld County, Colorado, by that of Philip, Cox, Fountain and Kilpatrick,[2, 3] of the United States Public Health Service. The presence of neutralizing antibodies in the serums of patients and horses indicated infection in some cases with one of these viruses, and in others with both. Similar evidence for mixed infection with the same viruses in human beings in California had previously been presented by Howitt.[6]

In the Yakima valley, the epidemics occurred during the two summers of 1939 and 1940. As might be expected in an outbreak due to two different agents, certain epidemiologic and clinical features were suggestive of each of the two types of encephalitis, although the general picture was more comparable to that of the St. Louis epidemic of 1933 than to any of the known equine-encephalomyelitis epidemics. Most of the patients lived near irrigation ditches or other potential sites of mosquito breeding. The possibility of insect vectors was not further investigated, but probable reservoirs for one or both viruses were suggested by the finding of neutralizing antibodies in the blood serums of a certain number of wild and domestic birds and mammals.

The reports from Weld County present similar

evidence of human infection with either one or both of these viruses. They further show that the serums of certain horses, not only from Colorado, but also from Montana and Washington, contained neutralizing antibodies for one or both viruses. The disease was reproduced experimentally in susceptible horses by the intracerebral inoculation of a proved strain of St. Louis virus. The resulting infection was indistinguishable clinically from that caused by the Western strain of equine-encephalomyelitis virus. It is thus evident that the differential diagnosis on clinical grounds is difficult, if not impossible.

Of even more interest, however, was the observation that during the course of this disease the infective agent was found in the nasal secretions but could not be detected in the blood. From the epidemiologic standpoint, therefore, it seems unlikely that transmission from the horse could be accomplished by blood-sucking insects. Moreover, since the virus is present in nasal secretions, transmission may be possible by direct contact, by vectors such as the common housefly or horsefly, by fomites, by water and so forth. These findings obviously provide a fertile field for further research.

More recently Hammon and his co-workers[7] have isolated the viruses of western equine and St. Louis encephalitis from mosquitoes (*Culex tarsalis*) collected from areas in which human encephalitis cases occurred. This evidence suggests the vector. Several species of wild and domestic birds and mammals may then be the important host-reservoirs,[4] whereas the horse remains only an accidental victim.

Effective therapy for these types of encephalitis is lacking. Specific therapeutic antiserums are of no avail at the present time because a sufficiently early etiologic diagnosis cannot be made. Prophylaxis with virus preparations, particularly for animals, offers some hope, but has not yet been investigated thoroughly enough. Particularly in view of the present defense program and the concentration of troops in camps, continued study of host reservoirs and methods of transmission is urgently needed to control these summer epidemics.

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*MAN ON HIS NATURE**

Sir Charles Sherrington, the dean of British physiologists, and long the chief exponent of structural neurology, whose life has been devoted to the elucidation of the reflex actions of the nervous system in its component parts and the integration of this action in the brain, was a particularly happy choice as Gifford Lecturer at Edinburgh in 1937–1938. Who better than Sherrington could, to quote his own words, use natural science to "provide a frame of reference for Natural Theology"? With the outlook of a poet, he provides science's generalizations about nature, using as a comparison the ideas on the same subject of Jean Fernel, a French physician of the midsixteenth century, who wrote *On Hidden Causes* (Paris, 1542), the first modern textbook of physiology.

Sherrington first points out the great changes in science that have occurred in the four hundred years since Fernel's time. Dealing with hard facts, the mechanical behavior of electrons, for example, we hardly talk the same language or think and research with the same tools. Science still, however, deals with facts, and facts only—smaller and smaller, to be sure, but still concrete. We can look at nature only with them in mind, but since man is part of nature, we can also look at man, although with slight understanding. We see brain, cells and processes, and even diagrammatically satisfy ourselves regarding reflex action. But how can we envisage "mind"? Energy does not seem to answer the question. Sher-

*Sherrington, C. S. *Man on His Nature*. The Gifford Lectures, Edinburgh, 1937–1938. 413 pp. New York: The Macmillan Company, 1941.

rington concludes that "mind" for anything ". . . perception can compass goes therefore in our spatial world more ghostly than a ghost. Invisible, intangible, it is a thing not ever of outline; it is not a 'thing.' It remains without sensual confirmation, and remains without it for ever. Stripped to nakedness there remains to it but itself. What then does that amount to? All that counts in life. Desire, zest, truth, love, knowledge, 'values,' and, seeking metaphor to eke out expression, hell's depth and heaven's utmost height." To this concept of "mind," Sherrington adds "the perceived world. . . . Together they make up the sum total for us: they are all we have."

This, and much more, will be found in this thought-provoking book, a scholarly treatise such as is rarely given to the world. Not by any means easy reading, it will provide rich nourishment, if slowly digested. In a busy, war-torn world, too few will have time for such an intellectual treat; but *Man on His Nature* will last, and no doubt will be read four hundred years from now, just as Sherrington reads Fernel's *On Hidden Causes*.

MEDICAL EPONYM

Korsakow's Syndrome

Sergei S. Korsakow (1853–1900), privatdocent of the Imperial University in Moscow, described the syndrome that bears his name in an article entitled "Ueber eine besondere Form psychischer Störung, combinirt mit multipler Neuritis (A Peculiar Form of Psychic Disturbance Associated with Multiple Neuritis)" in the *Archiv für Psychiatrie und Nervenkrankheiten* (21: 669–704, 1889). He refers to his first report on the subject, which appeared in a Russian journal, *Westnik Psychiatrii*, during 1887 under the title, "Disturbances in the Psychic Sphere Occurring in Alcoholic Paralysis, and Their Relation to the Psychic Disturbances in Multiple Neuritis of Nonalcoholic Origin." A portion of the translation follows:

> This psychic disturbance is shown sometimes in the form of a well-marked irritable weakness of the psyche, sometimes in the form of confusion, with quite characteristic disorientation in regard to place, time and situation, again as an almost pure variety of acute amnesia, with the most extreme sort of disturbance in the memory for recent events, while remote occurrences are well remembered. This unique psychic change is almost constantly present to a greater or less degree in the multiple neuritis of alcoholic patients; it is not, however, an exclusive characteristic of alcoholic neuritis, but also occurs in neuritides due to a variety of other causes.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY
SECTION OF OBSTETRICS AND GYNECOLOGY*

Fatal Shock, Following Placenta Previa and Massive Hemorrhage

A thirty-six-year-old para X entered the hospital, when approximately seven months pregnant, because of a sudden massive hemorrhage.

The patient had eight living children, and there had been one miscarriage. The last pregnancy was terminated by a cesarean section, and a living child weighing 9 pounds, 9 ounces, was delivered. The section was indicated because of an amputated cervix, and the repair of a third-degree tear of the perineum.

The patient had bled a little several times during the previous two months. Two weeks before admission, she had bled sufficiently so that a diagnosis of placenta previa was considered, although no vaginal examination was made. An x-ray film taken for the purpose of diagnosis did not show a placenta previa. On the day of admission, the patient had a very sudden massive hemorrhage at home and was sent to a hospital, where a vaginal pack was inserted. From this hospital, she was transferred to another institution, where the first pack was removed and a second introduced; a central placenta previa was found. Because the bleeding was excessive, cesarean section was performed immediately. A living child was delivered but died shortly after birth. The patient died four and a half hours after operation. The patient was apparently not transfused; the record states that she died of shock.

Comment. From the obstetric point of view, this patient was not ideally handled, and the fatal result should have been averted. Any patient who bleeds in the last trimester is entitled to an intelligent search for the cause of the bleeding. No intelligent diagnosis can be made without a vaginal examination. This patient was seen several times because of this bleeding, but at no time was a vaginal examination made. Had one been made and the cervix investigated, the central placenta previa would have been diagnosed, and the operation might have been done at election, not when the patient was weakened by excessive hemorrhage

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

and in moderate shock. X-ray study in the diagnosis of placenta previa is not always reliable; it should never take the place of a thorough and intelligent vaginal investigation.

The previous cesarean section made a second section imperative. No patient is so badly off that transfusion before operation is not of inestimable value, because any patient who succumbs while being transfused certainly would not survive, no matter what was done to her. It is barely possible that immediate transfusion, followed by operation, might have tided her over, but this is only problematical.

The lesson to be learned from this case is that all patients with bleeding in the last three months of pregnancy should be hospitalized, a definite diagnosis of the cause of bleeding made by vaginal examination, even at the expense of invading the cervical canal, and subsequent treatment based on this diagnosis given at the time of election. It is routine in most obstetric institutions that, when a vaginal examination is attempted on bleeding cases of this sort, a bagging kit and a laparotomy kit are both in readiness and an acceptable donor is at hand. It is only by following this technic that such fatalities can be averted.

DEATHS

BURNS—HIRAM H. BURNS, M.D., of Plymouth, died May 10. He was in his eighty-sixth year.

Dr. Burns received his degree from the Harvard Medical School in 1887 and was a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow survives him.

KRANTZ—MICHAEL KRANTZ, M.D., of Quincy, died September 4. He was in his sixtieth year.

Born in Russia, he received his degree from Middlesex University School of Medicine in 1921. He was a fellow of the Massachusetts Medical Society and of the American Medical Association.

His widow survives him.

MURPHY—DANIEL D. MURPHY, M.D., of Amesbury, died August 8. He was in his seventy-eighth year.

Dr. Murphy received his degree from the Dartmouth Medical School in 1891. At the time of his death, he was on the staffs of the Anna Jaques Hospital, Newburyport, and the Amesbury Hospital. He was a former president of the Essex North District Medical Society, and was a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow and two sons survive him.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR AUGUST, 1941

DISEASES	AUGUST 1941	AUGUST 1940	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	45	9	36
Chicken pox	125	81	105
Diphtheria	6	12	14
Dog bite	1043	1168	1112
Dysentery, bacillary	30	15	14
German measles	31	20	28
Gonorrhea	283	366	457
Measles	282	506	277
Meningitis, meningococcal	4	3	5
Meningitis, other forms	4		
Mumps	274	157	137
Paratyphoid fever	8	36	17
Pneumonia, lobar	74	91	110
Scarlet fever	196	77	110
Syphilis	341	297	415
Tuberculosis, pulmonary	281	298	301
Tuberculosis, other forms	26	25	31
Typhoid fever	9	11	11
Undulant fever	13	1	4
Whooping cough	575	508	511

*Based on figures for preceding five years.

GEOGRAPHICAL DISTRIBUTION OF CERTAIN DISEASES

Actinomycosis was reported from: Malden, 1; total, 1.

Anterior poliomyelitis was reported from: Beverly, 1; Boston, 4; Brockton, 3; Cambridge, 4; Dudley, 1; Leominster, 1; Fairhaven, 1; Haverhill, 2; Lawrence, 1; Lynn, 2; Malden, 1; Melrose, 1; Methuen, 5; New Bedford, 9; Northbridge, 1; Provincetown, 1; Quincy, 1; Somerville, 1; Taunton, 1; Uxbridge, 1; Wareham, 2; Worcester, 1; total, 45.

Anthrax was reported from: Peabody, 1; total, 1.

Diphtheria was reported from: Fall River, 5; Wrentham, 1; total, 6.

Dysentery, amebic, was reported from: Fitchburg, 1; total, 1.

Dysentery, bacillary, was reported from: Boston, 1; Braintree, 1; Cambridge, 1; Danvers, 7; Ipswich, 1; Melrose, 1; Medford, 1; Stoneham, 1; Watertown, 1; Worcester, 15; total, 30.

Infectious encephalitis was reported from: Cambridge, 1; Fall River, 1; New Bedford, 1; total, 3.

Malaria was reported from: Framingham, 1; total, 1.

Meningitis, meningococcal, was reported from: Cambridge, 1; Stoneham, 1; Wareham, 1; Yarmouth, 1; total, 4.

Meningitis, other forms, was reported from: Arlington, 1; Boston, 1; East Bridgewater, 1; Weymouth, 1; total, 4.

Paratyphoid fever was reported from: Beverly, 1; Cambridge, 1; Fort Banks, 1; Ipswich, 1; Lynn, 1; Medford, 2; Peabody, 1; total, 8.

Pellagra was reported from: Boston, 1; Peabody, 1; total, 2.

Septic sore throat was reported from: Boston, 1; Cambridge, 1; Greenfield, 1; total, 3.

Tetanus was reported from: New Bedford, 1; total, 1.

Trachoma was reported from: Fall River, 1; Natick, 1; total, 2.

Trichinosis was reported from: Boston, 1; Worcester, 1; total, 2.

Typhoid fever was reported from: Ayer, 1; Boston, 4; Deerfield, 1; Fall River, 1; Marblehead, 1; North Adams, 1; total, 9.

Undulant fever was reported from: Brookfield, 1; Deerfield, 1; Falmouth, 1; Franklin, 1; Haverhill, 1; Hopkinton, 1; Leominster, 1; Medford, 1; North Adams, 1; Orange, 1; Sheffield, 1; Uxbridge, 1; Worcester, 1; total, 13.

For the sixth consecutive year anterior poliomyelitis failed to reach epidemic proportions by the end of August.

Undulant fever was at a high level during the month.

Mumps and meningococcal meningitis both tended toward higher levels, though they have shown the usual seasonal decline.

Chicken pox, bacillary dysentery, scarlet fever and whooping cough were slightly above the five-year average.

Diphtheria, lobar pneumonia and typhoid fever were at unusually low levels.

Dog bite, gonorrhea, paratyphoid infection, syphilis and pulmonary tuberculosis were reported below the five-year averages.

Measles and German measles, which showed increased prevalences earlier in the year, made a rapid seasonal decline.

X-RAYING MILITARY MEN

Experience in World War I showed the value of discovering tuberculosis among military men. When the Selective Service Act went into effect, the Navy was requiring a chest roentgenogram for all enlisted and commissioned men, and the Army for the commissioned personnel only; facilities for routine roentgenography of all men were not at first available. Among the civilian agencies that supplemented the efforts of the Army in this emergency was the Bureau of Tuberculosis of the New York City Department of Health. These abstracts are taken from a record of that organization's experiences (Edwards, H. R., and Ehrlich, D. Examinations for tuberculosis, *J. A. M. A.* **117**:40–45, 1941).

An order issued October 28, 1940, by the Adjutant General's Office of the United States Army made it possible for civilian organizations to set up a roentgenographic service for men inducted into the Army. It provided for payment for x-ray films and for the services of civilian roentgenologists (under due control) until such time as the Army could assemble its equipment and assume full responsibility.

The Bureau of Tuberculosis of the New York City Department of Health has been engaged in mass roentgen-ray surveys of the apparently healthy population since 1933. These surveys have been accepted as a basic part of the tuberculosis-control program of New York City, and thus interest, based on experience, in providing a similar service for inductees and members of the State National Guard was keen. Accordingly, the bureau's mass roentgen-ray services, which were made possible through the WPA, were offered to the Surgeons of the Second Corps Area prior to the Adjutant General's order. Financial assistance was received from the tuberculosis associations of Queens and the Bronx.

After January 1, 1941, the Army assumed full financial responsibility for the roentgen-ray service in induction centers. The Department provided personnel for the interpretation of films. Since January 15, this service has also been taken over by the Army, which has assigned medical reserve officers qualified in this special field. The roentgenographing of National Guardsmen has been entirely at the expense of the Department of Health. Under existing regulations, the Army could not pay for this service until after induction, and it was essential that rejections be made before induction.

At the outset, there were four induction stations. Since January 1, 1941, all work has been done in two stations, one in Manhattan and one in Queens.

Those rejected men who were residents of New York City were given an appointment within the next two or three days to appear at the Health Department's Central Chest Clinic, where a complete study of the case was made. If this examination proved the original findings to be of no significance, the local draft board was so notified.

Rapid roentgenographic service was necessary, since the recruit was supposed to be cleared through all examinations by 2:30 p.m. of the day he reported at the induction station. With from 60 to 300 men per unit to be handled daily, even the rapid roll method used in the routine survey program was inadequate. Consequently, a special type of apparatus was devised. A modification of the roll paper camera was used in connection with a specially constructed portable dark room measuring 8 by 8 feet, with the back of the camera integrated into one side of the dark room. A signal device was installed between the roentgen-ray technician and the dark room. As soon as a film was exposed, the signal was flashed, and the dark room crew cut off the film and placed it in the developing bath. The signal was then reversed, indicating that another film was ready to be exposed. A team of three, consisting of a technician and two dark-room assistants, were able to operate faster than one exposure a minute. The films were processed in large trays and from the fixing bath were passed out to the physician through a light-proof pass. After being read, the films were washed in a portable tank and dried in a special device designed for the purpose.

Acceptance or rejection was based on Army regulations. Men showing any form of reinfection types of tuberculosis were rejected, because lesions of such types may become aggravated under conditions of military service. Primary lesions considered as active or extensive calcifications were likewise cause for rejection. Other forms of significant pulmonary disease, such as bronchiectasis, pneumonitis, atelectasis and extensive pleural changes, were cause for rejection until further study could determine their importance. Men with obviously abnormal cardiac silhouettes were reported to the medical examiners for such further study as might be indicated. Men with nothing more than apical caps, and those with small well-healed primary lesions were not rejected.

The group of men examined up to January 15, 1941, a period in which the Department of Health was actively engaged in the program, included 6609 inductees and 9541 guardsmen, a total of 16,150 who were x-rayed.

Of the inductees, 1.36 per cent were rejected and of the guardsmen, 1.21 per cent. About one third of the guardsmen were below the age of twenty-one, whereas only about 0.5 per cent of the inductees were below that age. An all-Negro regiment (National Guard unit) had the highest mean age in all groups and the highest rate of rejection, which was almost entirely on the basis of pulmonary tuberculosis. If the findings in this unit are subtracted from the totals of all National Guard units, a greater difference will be found between guardsmen and inductees.

Classification by stages of disease of the 70 men whose lesions were considered clinically significant shows that 65.7 per cent were minimal, 32.9 per cent moderately advanced and 1.4 per cent far advanced. Primary lesions indicated by calcific deposits were found in 6 per cent of the white men, 8.7 per cent of the Negroes and 7.1 per cent of the Puerto Ricans.

The group of men examined since January 16 and through March 31, 1941, totaled 35,210 men. During that period, the Department of Health's part has been

to re-examine and classify New York City men rejected at the induction center. In this time, 458 men have been rejected, 379 of whom have thus far been cleared at the Health Department Clinic. In 49, or 12.9 per cent, of those re-examined, the cause for rejection at the induction station was not confirmed, and the man was therefore considered suitable to be accepted in the Army.

A detailed cost analysis of personnel, equipment and materials necessary to complete this study indicated a total of $23,614.20. Using this as a basis for computation, the unit cost to examine each man by roentgenogram was $1.47. (The cost of taking a roentgenogram and its interpretation without any further follow-up was $13,911.20, or 58.8 per cent of the total.) The unit cost of rejecting a man for military service on the basis of the total cost was $106.02 for inductees and $122.37 for guardsmen.

Spillman has reported that the cost to the federal government of accepting a person with tuberculosis into the armed service is $10,000. Thus, in these studies involving 41,819 inductees and 9541 guardsmen, or a total of 41,360 men, 561 persons with chronic pulmonary tuberculosis were rejected, representing an estimated saving to the government of $5,610,000. — Reprinted from *Tuberculosis Abstracts*, October, 1941.

NOTES

The following appointments to the teaching and research staff of the Harvard Medical School were recently announced by the University (effective this year): Norbert A. Wilhelm, M.D., St. Louis University School of Medicine '25, lecturer on public-health practice; William V. Knoll, M.D., College of Medical Evangelists '35, Lucius N. Littauer Fellow in Pathology at the Collis P. Huntington Memorial Hospital; and Edward J. Welch, M.D., Harvard '36, assistant medical adviser.

Dr. Leon K. Baldauf has been appointed to a full-time professorship as head of the Department of Pathology at the School of Medicine at Middlesex University. He was formerly assistant in pathology at the Albany Medical College, resident pathologist at St. Louis City Hospital and professor of pathology and bacteriology at the University of Louisville School of Medicine; for the last three years, Dr. Baldauf has served as pathologist at the Cambridge City Hospital.

CORRESPONDENCE

PREMARITAL MEDICAL EXAMINATIONS

To the Editor: In reading the October 2 issue of the *Journal*, I came across Dr. Allen Johnson's remarks concerning premarital examinations on those persons who for several months or years have been traveling in the clouds of that grand passion and for the first time are about to put their feet once more on solid ground. What a rude awakening for the prospective groom to have a gloved finger roughly rammed through a spastic sphincter to massage a prostate in search of evidence of his past indiscretions.

Frankly, I believe there are a lot of us who are pretty much in the dark as to how far such an examination should be carried. Should it rest with a routine sample of blood or should smears also be taken? I think it would be of great help if there were some discussion of this in the *Journal*, and possibly a printed outline covering the essentials of such an examination in both men and women. Possibly some short form of examination sheet could be devised that could be incorporated with the regular history form; this would give uniform chartings throughout the State instead of a hit-or-miss sort of affair. Furthermore, if a patient could be shown such a blank and told "this is the regular form sponsored and approved by the Massachusetts Medical Society and used by every physician in the State," I do not believe there would be much antagonism to taking of general smears. The man or woman would merely think, "All the others have to go through the same procedure, so why should I complain?" I think this would greatly lessen the embarrassment of both parties, give a truer picture of the possibility of genitoinfectious disease and tide the doctor through the necessary procedures. I believe that such a slip of paper would well prove its worth to all concerned.

T. H. McSweeney, M.D.

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BOOK REVIEWS

Shock: Blood studies as a guide to therapy. By John Scudder, M.D., Med.Sc.D. 4°, cloth, 323 pp. with 55 illustrations and 5 plates, 3 in color. Philadelphia: J. B. Lippincott Company, 1940. $5.50.

This volume reviews the whole subject of shock and advances as the author's special contribution the theory that the increase in the potassium content of the plasma observed in shock (as in most cases of tissue injury) is in fact an evidence of potassium poisoning that plays a part in causing the shock syndrome, and the recommendation of adrenocortical hormone (Eschatin) as an important chemotherapeutic measure — based on the belief that it tends to restore blood volume and relieve hemoconcentration, to raise blood pressure, to combat decrease in alkali reserve by aiding the retention of bases and to promote excretion of potassium and ammonia by the kidney, and that it accomplishes a number of other desirable things. The reader must decide for himself to what degree these beliefs are substantiated. Concerning potassium, it is clear that the diminution of effective blood volume, which is admittedly an underlying cause of the shock syndrome, precedes the hyperpotassemia; there appears to be no evidence of an adrenocortical insufficiency in shock, although the use of the extract by the author in connection with other standard measures seems to have had good results.

Section I gives a historical introduction in three pages; Section II, on the theories of shock, is an enumeration of authorities supporting or opposing the responsibility of toxemia, the loss of circulating fluid, neurogenic dysfunction and adrenal exhaustion; Section III discusses blood changes in shock; Section IV, in discussing potassium, gives elaborate tables of lethal doses and seven pages of electrocardiograms in the experimental animal; Section V is entitled "The Historical Treatment of Shock" — meaning presumably the history of the treatment of shock. The next four sections deal briefly with physical measures of hemoconcentration, experimental shock, normal and abnormal blood values in the human subject and limiting factors in potassium analysis. Section X, although entitled "Advances in the Treatment of Shock," treats solely the use of the adrenocortical hormone, which is disposed of in five paragraphs, notwithstanding its importance as the author's main theme.

Sections XI and XII give adequate protocols of a number of cases illustrating various types of shock. The reader notes with interest the treatment followed, which in most cases includes the standard and the newer methods, and he may or may not agree with the author's enthusiasm for the use of the adrenocortical hormone. Section XIV,

entitled "Historical Development in the Conception and Treatment of Shock," lists two hundred and twenty-nine names and endeavors, in a brief sentence or two, to indicate the part played by each: for example, "Cannon, 1923, against adrenal glands in shock." Sections XV and XVI attempt to cover, respectively, in the same way, "The Physiologic and Toxicologic Effects of Potassium" and "Some of the Functions of the Adrenal Glands." Section XVII, which is a bibliography of five hundred and thirty-three references, is followed by author and subject indexes and a useful ten-page laboratory manual.

The volume is a sumptuous one characterized by lavishness in paper, illustrations, charts and symbolic graphs. Many pages contain but a short paragraph or a small chart. There are thirty blank pages for personal notes by the reader. Three beautiful full-page colored illustrations of test tubes, flasks and the normal adrenal gland add nothing that could not be satisfactorily dealt with by a few lines of text. In general, the author presents a wealth of brief citation without the all-essential critical evaluation and integration that would add so much to its value.

In clinical problems, the success of treatment is often proportional to the exactitude of available data in diagnosis. Dr. Scudder does well to remind the reader that the pulse rate and quality, the blood pressures, the blood count and the hemoglobin estimation are occasionally misleading and that the hematocrit, the specific gravity of blood and plasma and the level of plasma proteins add important evidence of hemoconcentration and diminished effective blood volume, which may permit suspicion of impending shock before other evidence exists. In spite of his assurances of the simplicity and ease of these determinations, one cannot help believing that they may add difficulty, delay and even confusion in a situation demanding prompt action. The reviewer believes that in most cases the experienced surgeon who first shrewdly evaluates the facts and circumstances of the trauma and then considers the patient as a whole will rarely have difficulty in diagnosing impending or existing shock, and will meet all indications for treatment if he adopts the almost universally accepted simple methods, including the creation and maintenance of an effective blood volume by the introduction of blood, plasma or, sometimes, other fluids that will remain in the vascular channels.

The Successful Error: A critical study of Freudian psychoanalysis. By Rudolf Allers, M.D., Ph.D. 8°, cloth, 266 pp. New York: Sheed and Ward, Incorporated, 1940. $3.00.

This is a useful book for those who wish to formulate their negative and critical reactions to psychoanalysis, and to those who have these feelings and wish to find them well expressed. Psychoanalysis is most actively promulgated by those who believe in it and those to whom it is a religion. They zealously defend it, but just as psychoanalysis has its adherents, it also has its critics, and Dr. Allers is one of these. The psychoanalyst will say that Dr. Allers does not understand psychoanalysis and that he is biased, hence not qualified to criticize it. In reading the book, one gets the impression that he has tried consciously to be objective, and certainly his criticism is based on his own philosophic and scientific training, which appears to be dignified. Dr. Allers has a medical background and training, and is probably better versed in the use of logic than most psychoanalysts. For these reasons and because his book is a thoughtful and sincere document, it is worth reading. Whatever the future may bring to prove the "rightness" or "wrongness" of psychoanalysis, this book will stand as one of the systematic and logical objections to numerous excessive claims for the therapeutic system with which Dr. Allers deals in detail.

One of the best chapters is on the subject of psychoanalysis and medicine. It is too complicated to summarize, but any physician, whether he is a psychoanalyst or not, will find that chapter interesting. If the reader is a psychoanalyst, he will probably find it very irritating.

This is not just another aimless dig or jab at psychoanalysis. It is a carefully leveled, well-aimed thrust.

Medical Progress: Annual, 1940. A series of fifty-two reports published during 1940 in the *New England Journal of Medicine.* Managing Editor: Robert N. Nye, M.D. 8°, cloth, 625 pp. Springfield, Illinois: Charles C Thomas, 1941. $4.00.

Each week in the *New England Journal of Medicine,* there appears a critical review of a definite subject by a man well qualified to evaluate the current progress in the field. These reviews are usually more than a mere report on the current literature, for they discuss the problems in general, particularly in relation to recent advances. The fifty-two articles published in 1940 have now been collected into a volume of great value to medical students, practitioners and specialists alike. The quality of the articles is extremely high, and the reviewer knows of no other annual publication that sets a finer standard. The papers are clearly written, and although they are scientifically accurate and often detailed in their exposition of the subject, the material is presented in such a way that the ordinary practitioner may read it with understanding. The articles contain bibliographies. The book should receive the widest recognition, and it is difficult to conceive of any doctor in the practice of medicine who would not improve his practice by reading part or all of this volume. For the specialist, moreover, who desires to keep up in fields other than his own, this book offers an invaluable opportunity. In addition, the book should be compulsory reading for all third-year and fourth-year medical students.

Necropsy: A guide for students of anatomic pathology. By Béla Halpert, M.D. 12°, cloth, 75 pp. St. Louis: The C. V. Mosby Company, 1941. $1.50.

This small book describes the autopsy technic practiced by Ghon and adopted by the author. The descriptions of technic are not always adequate because of the lack of illustrations. Its principal value lies in the suggestions of possible pathological findings in the various systems of the body.

NOTICES

MASSACHUSETTS MEDICAL BENEVOLENT SOCIETY

The annual meeting of the Massachusetts Medical Benevolent Society will be held at the Boston Medical Library on Monday, October 27, at 5:15 p.m. A meeting of the council will be held at 5 p.m.

MASSACHUSETTS MEMORIAL HOSPITALS

The regular staff meetings of the Massachusetts Memorial Hospitals will be held in the Evans Memorial Auditorium at 8:15 p.m. on October 31, January 2, February 27 and April 24. Dr. Chester S. Keefer will be the chairman. At the first meeting, Dr. Joseph F. Ross will speak on "The Metabolism of Radioactive Iron."

MASSACHUSETTS GENERAL HOSPITAL

The clinical staff meetings of the Children's Medical Service of the Massachusetts General Hospital were resumed on Friday, October 17, in the Ether Dome at 12 m. Subsequent meetings will be held on the following Fridays: October 31, November 14 and 28, December 12, and January 2, 16, and 30.

ROBERT BRECK BRIGHAM HOSPITAL

Dr. Carl C. Seltzer will speak on "Anthropometry and Arthritis: Difference between rheumatoid and degenerative joint diseases, males and females" at the Robert Breck Brigham Hospital on Monday, October 27, at 8 p.m. Drs. E. A. Hooten and Walter Bauer will lead the discussion.

Physicians and medical students are cordially invited.

PORTSMOUTH NAVAL HOSPITAL

There will be a meeting of the staff of the United States Naval Hospital, Portsmouth, New Hampshire, on Tuesday, November 4, at 8 p.m. Dr. Merrill Moore, of Boston, will speak on "Alcoholism: Its cause and treatment."

The members of the local societies in and about Portsmouth are cordially invited to attend.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Sunday, October 26

Monday, October 27

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

5:00 p.m. Massachusetts Medical Benevolent Society. Boston Medical Library, 8 Fenway.

*8 p.m. Anthropometry and Arthritis: Difference between rheumatoid and degenerative joint diseases, males and females. Dr. Carl C. Seltzer. Robert Breck Brigham Hospital.

Tuesday, October 28

*9:00–10:00 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital amphitheater.

5:00 p.m. The Chemical Reactions of the Body Fats. Prepared by Dr. Rudolf Schoenheimer and delivered by Professor Hans T. Clarke. Edward K. Dunham Lecture. Harvard Medical School amphitheater, Building C.

8:00 p.m. Massachusetts Society of X-Ray Technicians, Inc. New England Baptist Hospital.

Wednesday, October 29

New England Postgraduate Assembly. Sanders Theatre, Harvard University, Cambridge.

*9:00–10:00 a.m. X-ray Demonstration. Dr. Alice Ettinger. Joseph H. Pratt Diagnostic Hospital.

*12:00 m. Clinicopathological conference. Children's Hospital.

5:00 p.m. The Chemical Reactions of the Body Proteins. Prepared by Dr. Rudolf Schoenheimer and delivered by Professor Hans T. Clarke. Edward K. Dunham Lecture. Harvard Medical School amphitheater, Building C.

7:00 p.m. Neisserian Medical Society of Massachusetts. Hotel Kenmore, Boston.

Thursday, October 30

New England Postgraduate Assembly. Sanders Theatre. Harvard University, Cambridge.

*8:30 a.m. Combined clinic of the medical, surgical, orthopedic and pediatric services of the Children's Hospital and the Peter Bent Brigham Hospital, at the Children's Hospital. Conducted by Dr. F. R. Ober.

*9:00–10:00 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

3:00, 6:00 and 7:00 p.m. Massachusetts Public Health Association. Commander Hotel, Cambridge.

5:00 p.m. The Dynamic State of the Body Constituents. Prepared by Dr. Rudolf Schoenheimer and delivered by Professor Hans T. Clarke. Edward K. Dunham Lecture. Harvard Medical School amphitheater, Building C.

8:00 p.m. Some Local Uses of Estrogenic Hormones in Obstetrics and Gynecology. Dr. Norris W. Vaux. Journal Club meeting, Boston Lying-in Hospital.

Friday, October 31

*9:00–10:00 a.m. The Differential Diagnosis of Hemoglobinuria. Dr. T. Hale Ham. Joseph H. Pratt Diagnostic Hospital.

12 m. Clinical staff meeting of the Children's Medical Service. Massachusetts General Hospital Ether Dome.

8:15 p.m. The Metabolism of Radioactive Iron. Joseph F. Ross. Massachusetts Memorial Hospitals.

Saturday, November 1

*9:00–10:00 a.m. Presentation, with discussion, dispensary and district cases. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

October 29–November 1. Association of Military Surgeons. Page 473, issue of September 18.

October 31–January 30. Massachusetts General Hospital. Clinical staff meetings of the Children's Medical Service. Notice above.

October 31–April 24. Massachusetts Memorial Hospitals. Staff meetings. Page 672.

November 3–7. American College of Surgeons. Page vii, issue of July 31.

November 4. Portsmouth Naval Hospital. Notice above.

November 5–6. American Conference on Industrial Health. Page 473, issue of September 18.

November 13. Pentucket Association of Physicians.

November 17–19, 21–22, 27 and 30. Thomas William Salmon Memorial Lectures. Page 636, issue of October 16.

January 3. American Board of Obstetrics and Gynecology. Page 473, issue of September 18.

January 10–11. Forum on Allergy. Page 392, issue of September 4.

April 6–10. American Congress on Obstetrics and Gynecology. Page 600, issue of October 9.

April 20–24. American College of Physicians. Page 996, issue of June 5.

District Medical Societies

BERKSHIRE

October 30.
April 30.

BRISTOL NORTH

April 16. Taunton.

ESSEX NORTH

January 7. Haverhill.
May 6. Lawrence.

ESSEX SOUTH

November 12. Beverly Hospital, Beverly.
December 3. Salem Hospital, Salem.
January 7. Danvers State Hospital, Hathorne.
February 11. Lynn Hospital, Lynn.
March 4. Essex Sanatorium, Middleton.
April 1. Addison Gilbert Hospital, Gloucester.
May 13. Annual meeting (place to be announced).

FRANKLIN

November 11.
January 13.
March 10.
May 12. Annual meeting.

Meetings will be held at the Franklin County Hospital at 11:00 a.m.

HAMPSHIRE

November 5. Veterans Hospital, Leeds, 4:30 and 6:00 p.m.
January 7. Belchertown State Hospital, 1:00 and 4:00 p.m.
March 4. Hotel Northampton, Northampton, 4:30 and 6:30 p.m.
May 6. Hotel Northampton, Northampton, 8:30 p.m.

MIDDLESEX EAST

November 12.
January 28.
March 18.
May 6.

All meetings will be held at 12:15 p.m. at the Bear Hill Golf Club, Stoneham, except that of May 6, which will be held at Woburn at 6:30 p.m.

MIDDLESEX NORTH

October 29.
January 28.
April 29.

NORFOLK

October 28.
November 25.
January 27.
February 24.
March 24.
May 1–15 (date to be announced).

All meetings will be held at the Hotel Sheraton, Boston, except that of October 28, which will be held at the Marine Hospital, Brighton.

NORFOLK SOUTH

November 6.
December 4.
January 8.
February 5.
March 5.
April 2.
May 7.

All meetings will be held at 12:00 noon at the Norfolk County Hospital, South Braintree, with the exception of that of February 5, which will be held at the Quincy City Hospital, Quincy.

PLYMOUTH

November 20. Plymouth County Hospital, South Hanson.
January 15. Brockton Hospital, Brockton.
February 19. Jordan Hospital, Plymouth.
March 19. Goddard Hospital, Brockton.
April 16. Bridgewater State Farm, Bridgewater.
May 21. Lakeville Sanatorium, Middleboro.

SUFFOLK

April 29. Annual meeting. 8:15 p.m., Boston Medical Library.

WORCESTER

November 12. Grafton State Hospital, North Grafton.
December 10. Worcester City Hospital, Worcester.
January 14. St. Vincent Hospital, Worcester.
February 11. Worcester State Hospital, Worcester.
March 11. Memorial Hospital, Worcester.
April 8. Hahnemann Hospital, Worcester.
May 13. Annual meeting, Worcester Country Club, Worcester.

WORCESTER NORTH

January 28. Leominster Hospital, Leominster.
April 22. Burbank Hospital, Fitchburg.
July 22. Henry Heywood Memorial Hospital, Gardner.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Trauma and Disease. Edited by Leopold Brahdy, M.D., physician in charge of occupational diseases and injuries in the Office of the Corporation Counsel of the City of New York, and lecturer in radiology, New York University Medical School; and Samuel Kahn, M.D., medical examiner in the Bureau of Workmen's Compensation of the Department of Labor, State of New York. Second edition. 8°, cloth, 655 pp., with 13 illustrations and 13 tables. Philadelphia: Lea and Febiger, 1941. $7.50.

The Furtherance of Medical Research. By Alan Gregg, M.D., director for the medical sciences, Rockefeller Institute. 8°, cloth, 129 pp. New Haven: Yale University Press, 1941. $2.00.

Leaders of Medicine: Biographical sketches of outstanding American and European physicians. By Solomon R. Kagan, M.D. 8°, cloth, 176 pp., with 4 illustrations. Boston: The Medico-Historical Press, 1941. $3.00.

The Foot and Ankle: Their injuries, diseases, deformities and disabilities, with special application to military practice. By Philip Lewin, M.D., associate professor of bone and joint surgery, Northwestern University Medical School, professor of orthopedic surgery, Post-Graduate Medical School of Cook County Hospital, attending orthopedic surgeon, Cook County Hospital, attending orthopedic surgeon, Michael Reese Hospital, Chicago, and consulting orthopedic surgeon, Municipal Contagious Disease Hospital, Chicago. Second edition. 8°, cloth, 665 pp., with 304 illustrations. Philadelphia: Lea and Febiger, 1941. $9.00.

Nostradamus: The man who saw through time. By Lee McCann. 8°, cloth, 421 pp. New York: Creative Age Press, Incorporated, 1941. $2.75.

The New International Clinics: Original contributions, clinics, and evaluated reviews of current advances in the medical arts. Edited by George Morris Piersol, M.D., professor of medicine, Graduate School of Medicine, University of Pennsylvania, Philadelphia. Vol. III, N. S. 4, 1941. 8°, cloth, 300 pp. Philadelphia: J. B. Lippincott Company, 1941. $3.00.

The Roentgen Density of the Cystine Calculus: A roentgenographic and experimental study, including a comparison with more common uroliths. By Axel Renander. Translated from the Swedish by Catherine Djurklou. 8°, paper, 148 pp., with 67 illustrations and 23 tables. Stockholm: P. A. Norstedt and Sons, 1941. 15 Sw. Cr.

Essentials of General Surgery. By Wallace P. Ritchie, M.D., clinical assistant professor, Department of Surgery, University of Minnesota Medical School. 8°, cloth, 813 pp., with 237 illustrations. St. Louis: The C. V. Mosby Company, 1941. $8.50.

Bibliographia primatologica: A classified bibliography of primates other than man. Part I: Anatomy, embryology and quantitative morphology; physiology, pharmacology and psychobiology; primate phylogeny and miscellanea. By Theodore C. Ruch, B.Sc., Ph.D., Yale University School of Medicine. With an introduction by John F. Fulton, B.S., A.M., Ph.D., Yale University School of Medicine. 4°, cloth, 241 pp. Springfield, Illinois: Charles C Thomas, 1941. $8.50.

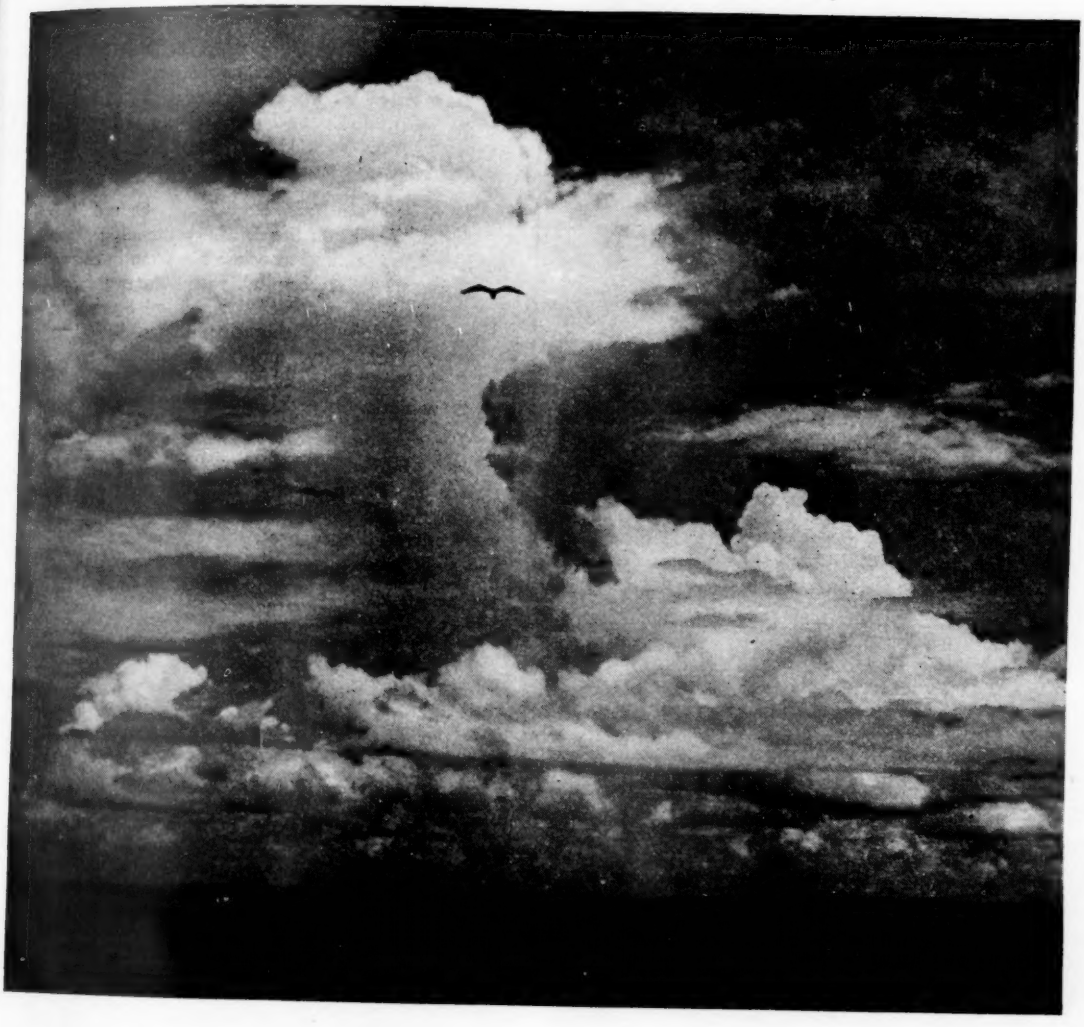
Sulfanilamide and Related Compounds in General Practice. By Wesley W. Spink, M.D., associate professor of medicine, University of Minnesota Medical School. 8°, cloth, 256 pp. Chicago: The Year Book Publishers, Incorporated, 1941. $3.00.

Society and Medical Progress. By Bernhard J. Stern. 8°, cloth, 264 pp. Princeton: Princeton University Press, 1941. $3.00.

A Manual of Bandaging, Strapping and Splinting. By Augustus Thorndike, Jr., M.D., associate in surgery, Harvard Medical School, and surgeon to the Department of Hygiene, Harvard University. 16°, paper, 144 pp., with 117 illustrations. Philadelphia: Lea and Febiger, 1941. $1.50.

Beiträge zur Röntgendiagnostik: Der Otitis Media Acuta und ihrer Komplikationen im Schläfenbein. By Silva

(Continued on page x)



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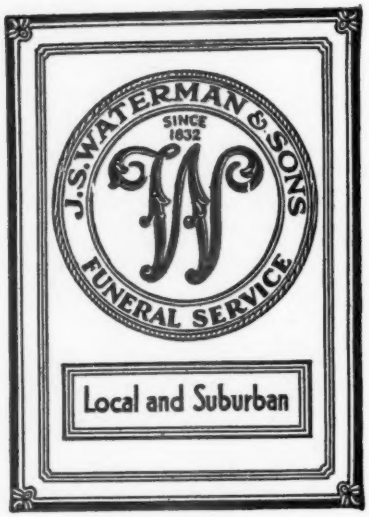

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Welin. 8°, paper, 180 pp., with 105 illustrations and 25 tables. Lund: Hakan Ohlssons Boktryckeri, 1941. 20 Sw. Cr.

Abdominal Surgery. Oxford Medical Outline Series. By John E. Hammett, M.D., professor of surgery, The New York Polyclinic Medical School and Hospitals, and attending surgeon, Coney Island Hospital, Department of Hospitals, New York City. 8°, cloth, 357 pp. New York: Oxford University Press, 1941. $2.00.

Diseases of the Respiratory Tract. Oxford Medical Outline Series. By Jacob Segal, M.D., attending physician, Riverside Hospital, and associate physician in charge of thoracic diseases Bronx Hospital, with a foreword by Harry Wessler, M.D., director of the Medical Service, Bronx Hospital, consultant in thoracic diseases, Beth Israel Hospital, New York City, and consultant in thoracic diseases, Beth Israel Hospital, Newark, N. J. 8°, cloth, 173 pp. New York: Oxford University Press, 1941. $2.00.

Fractures and Dislocations. Oxford Medical Outline Series. By Kenneth M. Lewis, M.D., assistant clinical professor of surgery, New York University College of Medicine, surgeon to the University Medical Office, Columbia University, adjunct professor of surgery, New York Polyclinic Medical School, associate visiting surgeon, Bellevue Hospital, and assistant visiting surgeon, Polyclinic Hospital. 8°, cloth, 218 pp. New York: Oxford University Press, 1941. $2.00.

Histology and Embryology. Oxford Medical Outline Series. By José F. Nonidez, Sc.D., professor of anatomy, Cornell University Medical College, New York City. 8°, cloth, 200 pp. New York: Oxford University Press, 1941. $2.00.

Obstetrics. Oxford Medical Outline Series. By Hervey Clock Williamson, M.D., associate clinical professor of obstetrics and gynecology, Cornell University Medical College, attending obstetrician and gynecologist, New York Lying-In Hospital, associate attending in obstetrics and gynecology, French Hospital, and consulting obstetrician, Bellevue Hospital, New York City, and Margaret Hague Hospital, Jersey City; and George Schaefer, M.D., assistant attending surgeon in Gynecology Clinic, St. Luke's Hospital, clinical assistant visiting obstetrician and gynecologist, Queens General Hospital, New York City, and assistant attending gynecologist, Sea View Hospital, Staten Island, New York. 8°, cloth, 114 pp. New York: Oxford University Press, 1941. $2.00.

Surgery of Head and Neck. Oxford Medical Outline Series. By Arthur S. McQuillan, M.D., clinical professor of surgery, New York University College of Medicine, surgeon to Bellevue, Misericordia, and Midtown hospitals, and consulting surgeon, New York Infirmary for Women and Children. 8°, cloth, 139 pp. New York: Oxford University Press, 1941. $2.00.

Thoracic Surgery. Oxford Medical Outline Series. By Charles W. Lester, M.D., assistant clinical professor of surgery, New York University College of Medicine, attending thoracic surgeon, Hospital for the Ruptured and Crippled, associate attending surgeon, Bellevue Hospital and New York Municipal Sanatorium, Otisville, assistant attending surgeon, Triboro Hospital, and consulting surgeon, Kings Park State Hospital. 8°, cloth, 141 pp. New York: Oxford University Press, 1941. $2.00.

Visual Outline of Psychiatry. Oxford Medical Outline Series. By Leland E. Hinsie, M.D., professor of psychiatry, College of Physicians and Surgeons, Columbia University, New York, and assistant director, New York State Psychiatric Institute and Hospital. 8°, cloth, 110 pp. New York: Oxford University Press, 1941. $2.00.